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Endovascular Therapy for the Carotid Blowout Syndrome in Head and Neck Surgical Patients: Diagnostic and Managerial Considerations

John C. Chaloupka, Christopher M. Putman, Martin J. Citardi, Douglas A. Ross, and Clarence T. Sasaki

PURPOSE: To review our institution's recent experience with patients with carotid blowout syndrome who were referred for emergency diagnostic angiography and endovascular therapy. **METHODS:** Eighteen consecutive patients who had had surgery for cancer of the head and neck and in whom carotid blowout syndrome had occurred were referred to our service in accordance with a standardized protocol. **RESULTS:** Twenty-three angiographic pathoetiologic conditions were diagnosed in the 18 patients; the majority of these were pseudoaneurysms involving various segments of the carotid system. Multiple lesions were detected in five patients. Most patients were treated by means of permanent balloon occlusion; in 8 patients with either multiple lesions or impending rupture requiring flap reconstruction, a *composite* permanent balloon occlusion of the affected carotid system was performed. Hyperacute hemorrhages were arrested in all cases. Hemorrhages reoccurred in 2 cases, and in 2 patients who had permanent balloon occlusion of the internal carotid artery, transient ischemic attacks occurred, which appeared to be related to temporary collateral reserve failure. No permanent neurologic complications ensued. **CONCLUSION:** Our recent experience with carotid blowout syndrome suggests that this clinical diagnosis represents a heterogeneous group of angiographic pathoetiologies that the physician should evaluate carefully before proceeding with endovascular therapy. Specific endovascular approaches depend on the pathoetiologic mechanism of active or impending hemorrhage and the urgency with which intervention is required.

Index terms: Arteries, carotid, internal; Interventional neuroradiology; Surgery, complications

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Rupture of the carotid artery, or so-called carotid blowout, is one of the most dreaded complications of head and neck surgery. It has been increasingly recognized as a delayed complication of aggressive primary and salvage radical neck surgery for extensive and/or recurrent squamous cell carcinoma of the head and neck (1). After the description of carotid blowout in 1962 by Borsany (2) and the report of a large

case series by Ketcham and Hoyer in 1965 (3), most studies have shown exceedingly high mortality and morbidity rates associated with this complication. Average estimates of cumulative mortality of approximately 40% and major neurologic morbidity of approximately 60% have been reported (1, 4-8).

A review of the literature reveals a variety of clinical presentations of actual and potential carotid rupture (1, 4, 5, 9-11), suggesting that so-called carotid blowout is more likely a clinical syndrome (ie, carotid blowout syndrome [CBS]), encompassing a spectrum of pathoetiologies and clinical manifestations (1).

Emergency surgical ligation (usually of the common carotid artery [CCA] or the proximal internal carotid artery [ICA] without provocative testing) has been traditionally the only therapeutic maneuver available for CBS, despite the well-documented risk of cerebral ischemia resulting in major neurologic morbidity and mor-

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tality (1–9). However, over the past several years, endovascular therapeutic techniques for occluding the major brachiocephalic arteries have been developed for the treatment of CBS (12–17). Although a few reports have documented the successful therapeutic occlusion of CBS with the use of these endovascular therapeutic techniques (9, 10, 16), the specific indications and technical approaches have not been well defined. To assess these issues better, we reviewed our institution's recent experience with patients with CBS referred for emergency diagnostic angiography and endovascular therapy.

Materials and Methods

Since 1993, our institution has implemented a standardized protocol for evaluating and treating patients with CBS. At our institution CBS is operationally defined as either an episode of acute hemorrhage (usually transoral or transcervical) or exposure of a portion of the carotid arterial system (usually from wound dehiscence or a devitalized musculocutaneous flap) in a patient who had previously undergone attempted curative or palliative surgical resection for a cervical carcinoma (usually squamous cell carcinoma). It has been our experience that the clinical spectrum of CBS can be subclassified into three groups (1). For the purposes of clarity and uniformity in describing these groups, we have developed a simple classification system that we have found useful for efficient communication and triage among the clinical services responsible for the care of these patients. We consider these group definitions to be equivalent to many previously described variations of CBS (1, 5, 10, 11).

The first group of patients with CBS includes those in whom there has been a breakdown of a wound from prior radical neck dissection or flap mobilization, resulting in a visibly exposed carotid artery. If this vessel is not promptly covered with healthy, well-vascularized tissue, it will almost inevitably rupture (1, 4, 18). Therefore, we describe this scenario as *threatened carotid blowout*.

A second commonly encountered group of patients with CBS are those with a short-lived acute hemorrhage that resolves either spontaneously or with simple surgical packing. The hemorrhage is usually either transoral or transcervical through a surgical wound or fistula. These episodes are frequently episodic and are considered sentinel hemorrhages stemming from a ruptured vessel with a pseudoaneurysm that leaks intermittently. Because there is no real wall with supporting structural elements around the pseudoaneurysm, complete rupture is a certainty and may occur at any time. We term this type of scenario *impending carotid blowout*.

The final group of patients with CBS are those who have an acute, profuse hemorrhage that is not self-limiting and is not well controlled with surgical packing. In this sce-

nario, there has been complete rupture of the affected artery, which is not confined by a pseudoaneurysm. These patients rapidly deteriorate from exsanguination unless immediate and intensive resuscitative measures and therapeutic occlusion of the ruptured artery are implemented. We define this clinical scenario as *acute carotid blowout*.

Between 1993 and 1995, 18 consecutive patients with a clinical diagnosis of CBS were referred to the interventional neuroradiology service for evaluation and treatment. All patients had undergone extensive primary and/or salvage radical resections and most (16 of 18) also had had adjuvant external-beam radiation. Patients with either *impending* or *acute* carotid blowout were treated with emergency carotid angiography after being aggressively resuscitated in the surgical intensive care unit with crystalloid, blood products, and pressers, as needed. All neuroangiographic and neurointerventional procedures were completed under intensive monitoring with the support of an anesthesiology team.

Standard neuroangiographic techniques were used in which high-resolution digital subtraction images (1024 × 1024 matrix) of the cervical and intracranial carotid circulation were obtained bilaterally. Meticulous attention to potential sites of endoluminal irregularity, disruption, pseudoaneurysm formation, and extravasation was focused on the distal CCA, the proximal external carotid artery (ECA), and the proximal cervical ICA. If bleeding occurred within the lower part of the neck, bilateral subclavian, costocervical, and thyrocervical artery angiography was also performed. If a causative lesion was detected that required permanent balloon occlusion of the ICA, angiographic evaluation of the circle of Willis was performed.

Endovascular therapy was guided primarily by angiographic findings in patients with either *impending* or *acute* CBS. Patients with *threatened* CBS were electively scheduled for diagnostic angiography and balloon test occlusion of the affected ICA. In both emergency and elective settings, if permanent balloon occlusion of the ICA was anticipated, a balloon test occlusion was attempted if the patient was hemodynamically stable and not bleeding profusely. A relatively standardized protocol in an awake patient was used, which has been described in detail in other publication (1, 19).

Therapeutic permanent balloon occlusion was performed only in cases of pseudoaneurysm formation or extravasation. The standard technique of occlusion of the carotid artery included use of detachable balloons (Goldvalve, Ingenor, France) mounted on microcatheters as described previously (15, 20). These balloons are not currently approved by the Food and Drug Administration for carotid occlusion, although exemptions for individual institutions for use of these devices are now available. If permanent balloon occlusion of the ICA was performed, at least two detachable balloons were positioned serially, inflated, and deployed to ensure permanent occlusion of the vessel (Fig 1). If only the ECA was considered the source of hemorrhage, then balloon occlusion of this vessel was performed by standard endovascular trapping, as de-



Fig 1. A, Right common carotid artery angiogram shows a subtle pseudoaneurysm of the cavernous internal carotid artery (ICA) (arrow).
 B, Lateral radiograph shows three detachable balloons used in permanent balloon occlusion of the right ICA. The most distal balloon covers the orifice of the pseudoaneurysm (arrow).

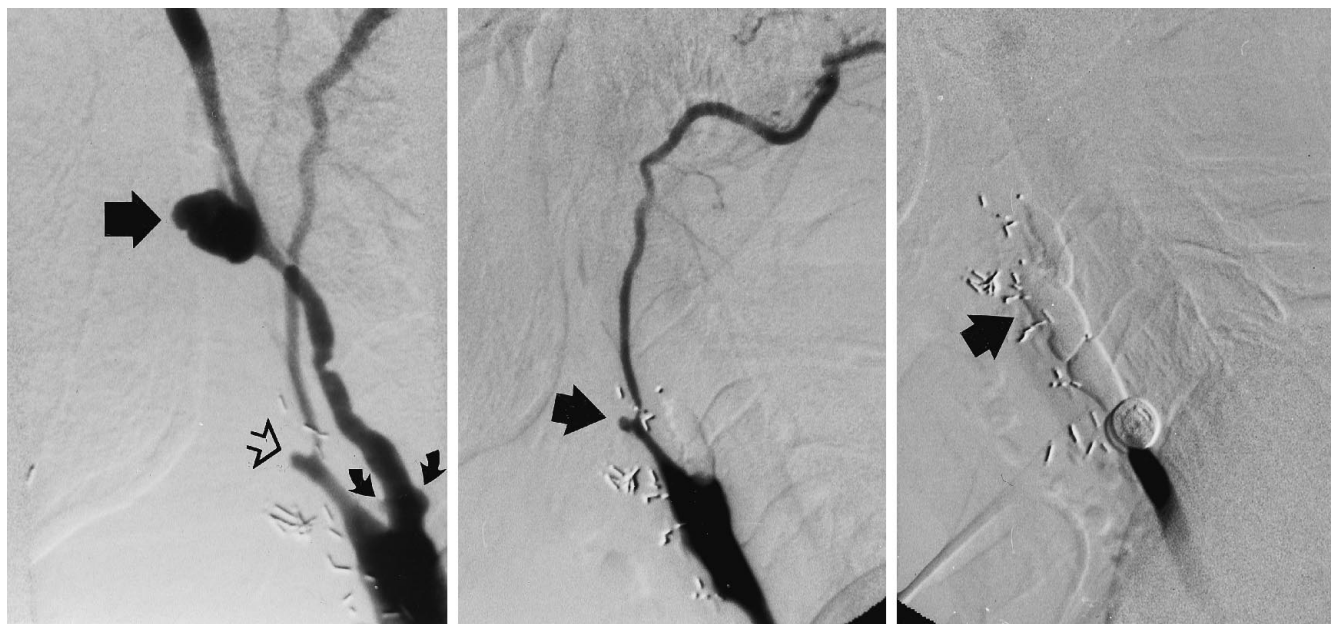


Fig 2. A, Right common carotid artery angiogram shows a large pseudoaneurysm of the mid-cervical right internal carotid artery (ICA) (large solid arrow). Small concurrent pseudoaneurysms of the proximal right ICA (curved arrows) and right external carotid artery (ECA) (open arrow) are also present.
 B, Right common carotid artery injection after balloon occlusion of right ICA shows the pseudoaneurysm of the right ECA (arrow) still fills.
 C, Angiogram obtained after composite permanent balloon occlusion shows embolization of the carotid system. A balloon has been positioned across the origin of the right ECA (arrow).

scribed previously (17). A composite permanent balloon occlusion of the ICA, ECA, and CCA was performed when lesions occurred near the origin of the ICA or within the CCA, or in cases of multiple lesions (usually involving a combination of the ICA and ECA) (Fig 2). Composite permanent occlusion was also performed empirically in all group 1 patients. The rationale for performing composite occlusion in this latter group was that these patients usually were candidates for salvage, en bloc surgical resection, in which complete occlusion of the carotid system facilitated such surgery (1).

In some situations, large-vessel therapeutic occlusion was not indicated (eg, in pseudoaneurysms of the distal ECA branches), and in these cases either platinum microcoils (Target Therapeutics, Fremont, Calif) or cyanoacrylate (Histoacryl, Braun, Germany) were used for embolic materials.

Following endovascular therapy, all patients were monitored in the neurointensive care unit. If permanent or composite occlusion of the ICA or of the carotid system was performed, the patient was anticoagulated with heparin for 48 hours and hydrated under close hemodynamic

TABLE 1: Clinical findings in patients with threatened carotid blowout

Case	Age, y/Sex	Clinical Diagnosis, Treatment	Angiographic Findings	Treatment	Complications	Outcome
2	68/M	SCCA, RND/EBRT/BT	ECA pseudoaneurysm at superior thyroidal artery	ECA embolization	None	Survived
8	64/M	SCCA, EBRT/BT, recurrence	R ICA encasement	Test and permanent occlusion of R ICA	None	Survived
12	79/M	SCCA, EBRT/BT	L ICA encasement	Test and permanent composite occlusion	TIA	Survived
15*	47/F	SCCA, RND, flap, wound dehiscence, EBRT Surgical exploration, exposed innominate artery	Irregularity of R ICA Occlusion of R CCA and inferior thyroidal artery	Test and permanent composite occlusion Permanent occlusion of innominate artery	None Balloon migration	Had recurrent hemorrhage Died (great vessel rupture)

Note.—BT indicates brachytherapy; CCA, common carotid artery; EBRT, external-beam radiation therapy; ECA, external carotid artery; ICA, internal carotid artery; RND, radical neck dissection; SCCA, squamous cell carcinoma; and TIA, transient ischemic attack.

* This patient was treated twice (see Table 3).

and neurologic monitoring. After discontinuance of heparin, all patients with ICA occlusion were placed on aspirin therapy. All patients were followed up until discharge from the hospital or death, typically for a period of 1 to 4 weeks.

Results

Twenty diagnostic and/or therapeutic procedures were performed in 18 consecutive patients who were referred to the interventional neuroradiologic service for evaluation and treatment of CBS. Relevant clinical data are summarized in Tables 1 to 3. Four patients had threatened CBS, 10 patients had impending CBS, and 5 patients had acute CBS (this included 1 patient who had had threatened CBS on two occasions). Twenty-three pathoetiologic conditions were identified, including 17 lesions seen on angiography. Eight of these were pseudoaneurysms of the ICA (35%), 6 were pseudoaneurysms of the ECA (26%), 1 was a pseudoaneurysm of the inferior thyroidal artery (4%), 1 was a pseudoaneurysm of the CCA (4%) (Fig 3), and 1 was tumoral hemorrhage (4%). Active extravasation was documented in only two cases (9%) (Fig 4). Pseudoaneurysms of the ECA were observed in the following locations: superior thyroidal artery (3 cases), dorsal lingual artery (1 case), ECA trunk (1 case), and buccal artery (1 case) (Figs 2 and 5). There were 5 cases of threatened CBS occurring in 4 patients. No angiographically definable lesion could be identified in 1 patient.

Permanent balloon occlusion was performed in 14 of 20 procedures. In 5 patients multiple lesions were detected, usually involving the ICA

and ECA trunk and/or branches (Figs 2 and 5). These patients and 3 others who had threatened carotid blowout were treated with composite permanent balloon occlusion of the affected carotid system (ie, of the ICA, ECA, and CCA) (Fig 2). Six single-vessel occlusions (4 of the ICA, 1 of the ECA, and 1 of the innominate artery) were also performed, usually to treat an isolated pseudoaneurysm (Fig 1).

In the group of patients who had permanent balloon occlusion, 12 of 14 had therapeutic occlusion of the ICA. Eight of these patients had previously been judged stable for preliminary balloon test occlusion, and all tolerated this provocative test well without neurologic deficits.

All patients with either impending or acute CBS had short-term arrest (first 48 hours) of their hemorrhages, although two patients had subsequent episodes of recurrent hemorrhage. One of these patients was subsequently found to have bleeding related to ulceration of a gastric pull-up, which was successfully treated. The other patient with recurrent hemorrhage had several episodes that finally led to her death caused by rupture of the root of the great vessels related to an uncontrolled infection of a musculocutaneous flap that extended into the mediastinum.

In two patients, mild transient ischemic attacks developed after permanent balloon occlusion of the ICA. The attacks appeared to be related to temporary collateral reserve failure. These patients responded to medical management (systemic anticoagulation, hypervolemia, and hemodilution) and had no permanent neu-

TABLE 2: Clinical findings in patients with impending carotid blowout

Case	Age, y/Sex	Clinical Diagnosis, Treatment	Angiographic Findings	Treatment	Complications	Outcome
1	57/M	SCCA, EBRT/BT	R CCA encasement	Test and permanent composite occlusion	None	Survived
3	63/M	SCCA, RND, flap	L ICA pseudoaneurysm	Permanent composite occlusion	None	Survived
6	60/M	SCCA, RND, EBRT/BT	ECA pseudoaneurysm at superior thyroidal artery	Permanent occlusion of ECA	None	Survived
7	79/F	SCCA, RND, EBRT/BT	L ICA and internal maxillary artery stenosis	Test and permanent occlusion of L ICA	None	Survived
10	70/F	SCCA, salvage resection, flap, EBRT/BT	L ECA pseudoaneurysm L ICA, pseudoaneurysm, L ECA encasement	Test and permanent composite occlusion	None	Survived
11	57/M	SCCA/EBRT/BT	R CCA pseudoaneurysm, L ICA encasement	Test and permanent composite occlusion	Femoral artery pseudoaneurysm	Died (retroperitoneal hemorrhage)
13	20/M	Ewings EBRT 2° fibrosarcoma	R ICA pseudoaneurysm	Test and permanent occlusion of R ICA	None	Survived
16	52/M	SCCA, EBRT, BT, CT-guided fine-needle biopsy	R ECA pseudoaneurysm at buccal artery	Superselective embolization	None	Survived
17	69/M	SCCA, flap, gastric pull-up, EBRT/BT	R ECA pseudoaneurysm at superior thyroidal artery	Superselective embolization	None	Had recurrent hemorrhage of R internal jugular vein; second recurrent hemorrhage, gastric necrosis, ulcer
18	69/F	SCCA/RND/EBRT/BT	99% stenosis of R CCA	None	None	Survived

Note.—BT indicates brachytherapy; CCA, common carotid artery; EBRT, external-beam radiation therapy; ECA, external carotid artery; ICA, internal carotid artery; RND, radical neck dissection; and SCCA, squamous cell carcinoma.

rologic complications or ipsilateral vision loss. Two patients died during their hospital stay. One patient with an underlying coagulopathy abruptly became hypotensive and died 1 week after occlusion of the ICA. Although an autopsy was not performed, this patient's death was presumed to have resulted from a massive retroperitoneal hemorrhage caused by rupture of a pseudoaneurysm of the femoral artery that was documented after an arterial sheath was removed. This patient's death was therefore considered related to a delayed technical complication. One patient died of rupture of the great vessels resulting from mediastinitis.

One additional technical complication occurred during permanent balloon occlusion of the innominate artery in which a balloon migrated and subsequently lodged in the right

common iliac artery. This balloon was successfully retrieved without sequelae.

Overall outcomes were good, with 16 (89%) of 18 patients surviving CBS. One fatality was probably related to a delayed technical complication. There was no major neurologic morbidity, but 2 (11%) of the 18 patients had recurrent hemorrhages, of which one was ultimately fatal.

Discussion

Rupture of the carotid artery, often resulting in catastrophic hemorrhage, is a well-known complication of aggressive surgical management of squamous cell carcinoma of the head and neck. During the course of therapy, the carotid system and/or its major branches occasionally become injured, resulting in weakening of the arterial wall that predisposes to rupture.

TABLE 3: Clinical findings in patients with acute carotid blowout

Case	Age, y/Sex	Clinical Diagnosis, Treatment	Angiographic Findings	Treatment	Complications	Outcome
4	57/M	SCCA, salvage resection, RND, EBRT, recurrence	Tumor neovascularity buccal and submental ECA	Superselective embolization with PVA	None	Survived
5	57/M	SCCA, EBRT, recurrence	R ICA encasement	Permanent occlusion of R ICA	TIA	Survived
9	63/M	SCCA, RND, EBXT/BT, recurrence	R ICA pseudoaneurysm with extravasation, R ECA pseudoaneurysm at dorsal lingual artery	Permanent composite occlusion	None	Survived
14	57/M	SCCA, RND, EBRT/BT, recurrence	L ECA occlusion and extravasation, L ICA stenosis with pseudoaneurysm	Test and permanent composite occlusion	None	Survived
15*	47/F	SCCA, RND, flap dehiscence, EBRT	Permanent balloon occlusion of ICA, pseudoaneurysm of inferior thyroidal artery	Superselective embolization of inferior thyroidal artery	None	Recurrent hemorrhage

Note.—BT indicates brachytherapy; EBRT, external-beam radiation therapy; ECA, external carotid artery; ICA, internal carotid artery; PVA, polyvinyl alcohol particles; RND, radical neck dissection; SCCA, squamous cell carcinoma; and TIA, transient ischemic attack.

* This patient initially had a threatened carotid blowout (see Table 1).

Fig 3. Angiogram shows a large pseudoaneurysm of the common carotid artery (*large arrow*). Narrowing of the internal carotid artery (*small arrows*) is also present, most likely related to postoperative cicatrization, radiation-induced changes, or tumor encasement.

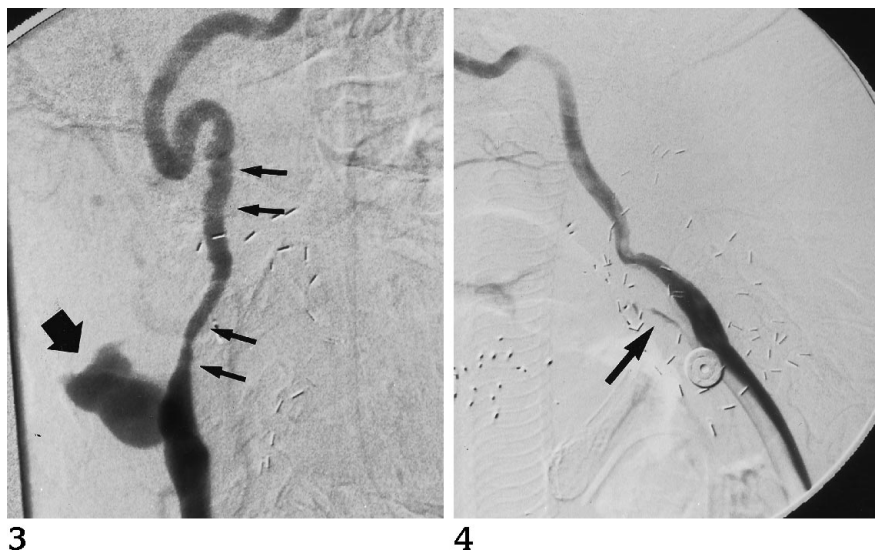


Fig 4. Left common carotid artery (CCA) angiogram shows a small amount of extravasation from the CCA into the oropharynx (*arrow*) from a fistula that developed after rupture of a pseudoaneurysm.

Radiation therapy (both external beam and brachytherapy), radical resection, flap necrosis with carotid exposure, wound infection, pharyngocutaneous fistula, and recurrent/persistent carcinoma all have been implicated as potential pathogenetic mechanisms (3–5, 8, 18, 21–25).

Although carotid rupture or so-called carotid blowout has been frequently described in the literature, there has been considerable variation in the way this clinical entity is defined and the specific use of terminology, which has led to

some confusion in comparing reported case series (1, 10, 11). Consequently, for the purposes of clarity and uniformity in describing these patients, we have developed a simple classification system that is based on the premise that carotid blowout is actually a clinical syndrome (ie, carotid blowout syndrome or CBS) with a well-defined spectrum of manifestations. The group definitions outlined in the Materials and Methods section (ie, *threatened*, *impending*, and *acute* CBS) are derived from some previ-

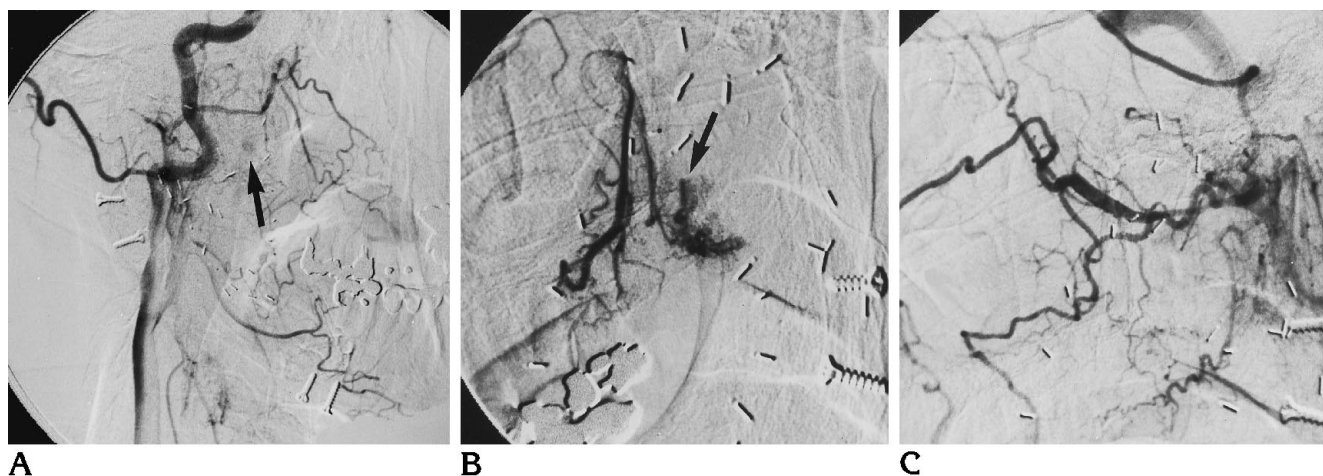


Fig 5. A, Right common carotid artery angiogram (late arterial phase) shows a subtle collection of contrast material in the masticator space, which persisted late into the venous phase (arrow).

B, Superselective catheterization of a buccal branch of the internal maxillary artery confirms a pseudoaneurysm (arrow).

C, External carotid artery injection after embolization of the buccal branch with cyanoacrylate shows occlusion of the pseudoaneurysm.

ously reported presentations and descriptions of carotid rupture within the literature, and are meant to facilitate both the general applicability and comparability of this classification system (1, 5, 10, 11).

Traditionally, emergency surgical ligation of the CCA or ICA has been used to treat CBS (2-8, 23-25). This approach, however, has generally resulted in an unacceptable rate of major complications, such as death and stroke. Cumulative data from these series indicate that the average estimated mortality is approximately 40% (varying from 9% to 64%) and major neurologic morbidity is approximately 60% (varying from 9% to 84%) (1, 4-8, 10, 23-25). Isolated series have reported even greater morbidity. For example, Maran et al reported 100% mortality in 17 patients with CBS (8).

Several general limitations of surgical management of patients with CBS may explain the associated high morbidity and mortality. Patients with CBS may be hemodynamically unstable, which greatly complicates perioperative management. General anesthesia can be complicated by profound hypotension in patients with depleted intravascular volumes, resulting in severe global cerebral ischemia. Extreme blood loss can produce a consumption coagulopathy, resulting in uncontrollable rehemorrhage. Extensive, often multiple, previous operations in combination with some form of radiation therapy can make surgical dissection extremely challenging.

Another important limitation of previous surgical series is that they were invariably performed without diagnostic angiography. Consequently, the type and precise location of the cause of hemorrhage was not always known, and often was simply inferred from the physical examination. Judging from our recent experience, it is likely that the former standard technique of common carotid ligation often did not adequately address the underlying cause of many hemorrhages associated with CBS.

An additional compounding problem with the usual surgical technique of proximal carotid ligation is the increased risk of thromboembolic complications resulting in stroke caused by the phenomenon of propagating thrombus within a long segment of the proximally occluded carotid system. Dandy (26, 27) correctly recognized that thromboembolic phenomena after carotid ligation were due to propagation of thrombus within the large intravascular dead space distal to the site of occlusion. Historically, extensive clinical experience with both open surgical and endovascular parent artery occlusion (mostly for treatment of giant intracranial aneurysms) has shown that proximal occlusions of the carotid artery within the neck are associated with a relatively high risk of thromboembolic stroke (19, 28). This risk of stroke from surgical ligation also is likely to have been increased in these surgical series by the lack of systemic anticoagulation.

Over the past two decades, advances in interventional neuroradiology have led to the development of endovascular therapeutic techniques for permanently occluding major blood vessels, such as the carotid artery. These developments have expanded the role of endovascular therapy in managing patients with various intracranial and extracranial diseases of the brachiocephalic vessels. In 1974, Serbinenko reported the application of detachable balloon catheters for the occlusion of cerebral blood vessels (12). Debrun et al later applied this approach to the treatment of carotid cavernous fistulas (14, 29). These techniques also can be used with great success for treating certain types of intracranial giant aneurysms (13, 15, 19, 20, 30, 31).

Application of these techniques to the cervical portion of the carotid system is a natural extension of endovascular therapy. The vascular injuries sustained by patients during the course of treatment of head and neck squamous cell carcinomas appear well suited to endovascular therapy, although its precise role in terms of diagnosis, indications, and specific technical approach remains poorly defined. In 1984, Osguthorpe and Hungerford reported the use of detachable balloons specifically for the treatment of impending carotid rupture in a patient with a cervical carcinoma (16). Subsequently, Sanders et al in 1986 reported a case of carotid rupture that was successfully treated with balloon embolization (10). One year later, Zimmerman et al published their findings from a small series of six patients treated with endovascular occlusion for so-called impending carotid rupture, in which successful therapeutic occlusion was achieved in all patients, although two patients required a carotid bypass procedure and one patient suffered a major stroke (11). Most recently, Citardi et al reported the results of a study of 10 patients treated with endovascular techniques among their series of 16 patients with CBS (1). In all 10 patients, acute hemorrhages were arrested without serious permanent neurologic sequelae.

Since 1989, all patients with CBS at our institution have been referred for diagnostic angiography. As in an earlier series reported from our institution by Citardi et al (1), we have found a surprisingly heterogeneous group of angiographic pathoetiologies of hemorrhage in patients with CBS. In the literature, most carotid ruptures have been reported to occur in the

CCA, with only approximately 15% identified in the ICA and none in the ECA (3, 6, 24). These results are in striking contrast to the 10 lesions in the ICA (5 pseudoaneurysms, 5 impending ruptures) (43%), 7 lesions in the ECA (30%), 2 lesions in the CCA (1 pseudoaneurysm, 1 impending rupture) (9%), 1 tumoral hemorrhage (4%), and 1 inferior thyroidal lesion (4%) found in our current series of 18 patients with 23 pathoetiologic conditions. Five patients in our series also had multiple concurrent lesions on angiography (Fig 2).

Because of this heterogeneity in angiographically identified pathogenetic lesions, we believe that meticulous angiographic evaluation is necessary both to plan for properly and to use judiciously endovascular therapeutic techniques. Without the use of selective and superselective catheterization and high-resolution digital subtraction angiography, many of the lesions observed in our series may have been missed, resulting in failure to address the underlying cause of hemorrhage. If an empiric therapeutic approach were adopted (eg, ICA occlusion), the patient could ultimately be exposed to excessive and unnecessary risks of treatments or fail to be protected from further hemorrhage.

By using the anatomic and pathologic information obtained from good-quality angiography, the interventional neuroradiologist can match the best endovascular technical approach(es) to the lesion responsible for the hemorrhage. Pseudoaneurysms involving the ICA, CCA, and proximal ECA trunk are best approached with detachable balloons, since these devices provide good control in positioning and deposition, usually permitting rapid and complete arterial occlusion upon inflation. If multiple pseudoaneurysms are detected in the carotid system, then we favor composite permanent balloon occlusion, in which the ICA, proximal ECA trunk, and distal two thirds of the CCA are therapeutically occluded. The composite occlusion allows for delayed carotid resection in candidates for salvage surgical management and minimizes the risk of a second carotid rupture at another site within the injured carotid system. An illustrative example is our case 10, a patient who had multiple pseudoaneurysms of the ICA, CCA, and ECA (Fig 2). In this case, if only the obvious right ICA pseudoaneurysm had been treated with endovascular trapping by permanent balloon occlusion, the

patient would have remained at substantial risk for additional hemorrhages.

Isolated pseudoaneurysms of the ECA can be approached by superselective catheterization of the involved vessel followed by transarterial embolization with coils or cyanoacrylate. For example, without superselective catheterization of the ECA and internal maxillary artery in case 16, the pseudoaneurysm of the buccal artery would not have been identified (Fig 5). In this case, empiric permanent balloon occlusion of the ICA or composite occlusion of the ipsilateral carotid system most likely would not have provided protection against further bleeding. In rare situations, hemorrhage associated with CBS may be due to bleeding from the tumor, in which case superselective catheterization and embolization of the feeding arteries with particulates could result in significant palliation.

Endovascular therapeutic approaches to CBS have several potential risks that are related both to technique and to the disease targeted for treatment. Most of the technique-related risks, such as thromboembolic and foreign body embolic stroke, are the same as those reported in prior series (12–15, 17). Reflex bradycardia and hypotension from stimulation of the carotid body occasionally may occur from mechanical manipulation of the carotid bulb when inflating a detachable balloon or from abrupt increases in pressure within the proximal carotid system after distal occlusion of the ICA.

An important additional risk of an endovascular approach to CBS is acute rupture of a pseudoaneurysm before or during inflation of a detachable balloon. This event can have catastrophic consequences, with rapid exsanguination and severe aspiration if adequate intensive supportive preparation and definitive treatment of the rupture is not provided.

Because of the risks associated with endovascular therapy for CBS, full intensive monitoring is essential. The best management of such cases includes placement of large-bore peripheral and central intravenous resuscitation lines, an arterial line, noninvasive oxygen saturation monitoring, and electrocardiographic monitoring. It has been our experience that this type of intensive monitoring to enable a rapid response to complications during endovascular therapy is best accomplished by a multidisciplinary approach, with the full participation of both an anesthesiology and head and neck surgical team.

Most of the patients in our series underwent therapeutic occlusion of the ICA. An important potential delayed complication of endovascular therapy for CBS is stroke, which may result from either hemodynamic insufficiency (eg, systemic hypotension or delayed collateral reserve failure) or thromboembolic phenomena (15, 17, 19). To minimize the risk of postocclusion thromboemboli, a distal occluding balloon is positioned within the petrous portion of the ICA whenever possible. In addition, our preliminary experience indicates that routine, short-term anticoagulation of these patients for 48 hours after permanent balloon occlusion reduces the risk of delayed thromboembolic events.

In an attempt to decrease the risk of both acute and delayed hemodynamic ischemia related to occlusion of the ICA, we also attempt to perform a conventional balloon test occlusion of the ICA in all patients with CBS who are being considered for ICA occlusion, since this provocative test has proved useful in identifying some patients who will not tolerate permanent occlusion of the ICA (17, 30, 31). Although it is well recognized that conventional balloon test occlusion will fail to identify a small subset of patients in whom delayed hemodynamic ischemia will develop after permanent balloon occlusion of the ICA, no additional adjunctive testing of cerebral blood flow was performed in our patients, since it was our opinion that they all were too unstable for such evaluation, and the potential benefits of such testing are questionable (1).

Although not encountered in our current series, the problem of patients who require therapeutic ICA occlusion who fail conventional balloon test occlusion also must be considered. The usual option of performing extracranial to intracranial bypass in such patients is usually not possible owing to a combination of the typically urgent nature of the situation and the technical limitations of performing this surgery in patients who have had radical neck surgery and radiation treatment. Currently, our approach to patients with CBS is to discuss with them and their families, before the test occlusion, the likely consequences of permanent occlusion of the ICA in terms of neurologic outcome if the test occlusion fails. It has been our experience that usually patients and their families will not consent to permanent ICA occlusion if the test occlusion results in a neurologic deficit. For the future, we are considering cov-

ered stent application to the diseased portions of the carotid system as an alternative endovascular treatment in patients with pseudoaneurysms of the CCA and ICA.

The outcomes in our series compare well with previous surgical series in which 89% of our patients survived CBS with no major neurologic morbidity related to therapy. The rate of delayed transient ischemia related to permanent occlusion of the ICA (2 [15%] of 13 patients) is comparable to that in previous reports (15, 31, 32). The death that occurred in a patient who abruptly became hypotensive 1 week after carotid occlusion may have represented a delayed technical complication caused by presumed retroperitoneal hemorrhage from a ruptured femoral pseudoaneurysm.

We conclude that endovascular therapy for the treatment of CBS in the context of a well-organized and coordinated multidisciplinary protocol is an excellent management approach, with a high rate of both technical and clinical success and an acceptable rate of complications.

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