The Journal of Trauma: Injury, Infection, and Critical Care
Issue: Volume 42(1), January 1997, pp 123-132

Publication Type: [Case Reports]
ISSN: 0022-5282
Accession: 00005373-199701000-00023

[Case Reports]

Gunshot Wounds of the Internal Carotid Artery at the Skull Base: Management with Vein Bypass Grafts and a Review of the Literature

Rostomily, Robert C. MD; Newell, David W. MD; Grady, M. Sean MD; Wallace, Sharon MD; Nicholls, Stephen MD; Winn, H. Richard MD

Author Information

From the Department of Neurological Surgery (R.C.R., D.W.N., M.S.G., H.R.W.), the Department of Surgery, Division of Vascular Surgery (S.N.), and Department of Radiology (S.W.), University of Washington School of Medicine, Harborview Medical Center, Seattle, Washington.

This work was supported in part by the following grants: NIH P50 NS30305 (M.S.G., D.W.N.), 1 K08 NS 01569 01 (D.W.N.), and NIH Clinical Neuroscience Training Program Grant 2T 32-NO7144 (R.C.R.).

Address for reprints: David W. Newell, MD, Department of Neurological Surgery, University of Washington School of Medicine, Harborview Medical Center, 325 Ninth Avenue, Box 359766, Seattle, WA 98104.

Abstract

Background: Penetrating trauma to the skull base and distal cervical internal carotid artery (ICA) can result in occlusion or pseudoaneurysm formation. The appropriate management strategy for these rare lesions is controversial and includes observation, anticoagulation, carotid ligation, balloon occlusion, or revascularization.

Methods: We present the management and outcomes of four consecutive patients, two with pseudoaneurysms and two with acute occlusions, after injury to the distal cervical/petrous ICA from gunshot wounds. Preoperative assessment determined intracranial collateral flow patterns and the patency of the distal portion of the petrous ICA.

Results: Two patients underwent cervical-to-petrous ICA vein bypass grafts without neurologic complications. Both grafts remain patent without evidence of emboli at 2 years and 3 months, respectively. Both of the conservatively managed patients died, one from a massive cerebral infarction and the other from intracerebral hemorrhage.

Conclusions: These cases underscore the need for an aggressive approach to the assessment and management of patients with penetrating vascular skull-base injuries. Although the optimal treatment of remains controversial, when the goal is exclusion of the injured portion of the carotid artery and revascularization, the cervical to petrous ICA vein bypass graft is a valuable management option that can reduce the potential morbility and mortality from acute ischemic or delayed embolic or hemorrhagic complications, provide immediate restoration of high flow, and allow good surgical access with minimal risk to intracranial structures.

Key Words: Bypass graft, Gunshot wound, Internal carotid artery, Skull base.

Penetrating trauma near the skull base is often associated with high cervical/proximal intracranial internal carotid artery (ICA) injuries. [1-6] In addition to transection, direct injury or secondary concussive forces can produce pseudoaneurysms and vessel dissection with or without occlusion. Treatment of these lesions is based on the potential for secondary neurologic deficits from hemorrhage or ischemic events. Surgical treatment options are limited by the difficulty inherent in achieving adequate direct access in this region and have led to the use of vascular bypass procedures to treat high cervical/petrous ICA lesions. The use of a cervical-to-petrous carotid artery reversed saphenous vein bypass graft (VBG) allows immediate high-volume flow without the need for intracranial exposure and dissection. In contrast, extracranial-to-intracranial (EC-IC) bypass provides lower volume flow and requires intracranial exposure and dissection.

In this report, we describe the management of four cases of gunshot wounds (GSWs) to the skull base that produced either traumatic pseudoaneurysms or ICA occlusions, including two patients who were successfully treated with cervical-to-petrous VBGs. Preoperative assessment, surgical treatment options, and considerations in patient selection are discussed. The potential catastrophic consequences of these injuries are also highlighted in the presentation of two patients who were managed conservatively.

MATERIALS AND METHODS

Preoperative Assessment

Four consecutive cases of patients with skull base GSWs and injury to the ICA treated at Harborview Medical Center, Seattle between March 1994 and June 1995 were selected for inclusion in this report. The initial preoperative assessment in each case included computed tomographic (CT)scans of the brain, including fine cuts through the skull base. A contrastenhanced CT scan with fine cuts was obtained in selected cases to provide anatomic information about the location and configuration of a traumatic aneurysm in the carotid canal. A complete cervical carotid and four-vessel cerebral angiogram was obtained in all cases. A full angiogram is necessary to establish whether the petrous portion of the ICA is patent and suitable for grafting and to define potential collateral circulation patterns. Other tests that were variably obtained as part of the preoperative assessment include single photon emission computed tomography (SPECT), transcranial Doppler (TCD), and carotid duplex examination. The studies obtained for each patient are outlined in the case reports.

Operative Technique

The operative technique of cervical-to-petrous ICA vein bypass used in the following cases has been described in detail elsewhere [7,8] and is illustrated in Figure 1 Briefly, the ICA is exposed for several centimeters distal to the bifurcation of the common carotid artery. Then a small temporal craniotomy is performed and the petrous carotid is exposed extradurally by drilling off the petrous bone, taking care to limit the posterior exposure to prevent entry into the cochlea. The greater superficial petrosal nerve is divided to prevent traction on the geniculate ganglion that might result in facial nerve injury. The patient is heparinized and then the petrous carotid artery is divided at the most proximal aspect of the petrous exposure and a reversed saphenous vein graft is then anastomosed to the distal portion of the petrous ICA. The graft is tunneled subcutaneously or along the course of the superficial temporal artery and anastomosed to the proximal cervical ICA. The graft patency can be monitored by Doppler ultrasonography before closure. Intraoperative monitoring with electroencephalography and TCD is reserved for patients with a patent ipsilateral ICA.

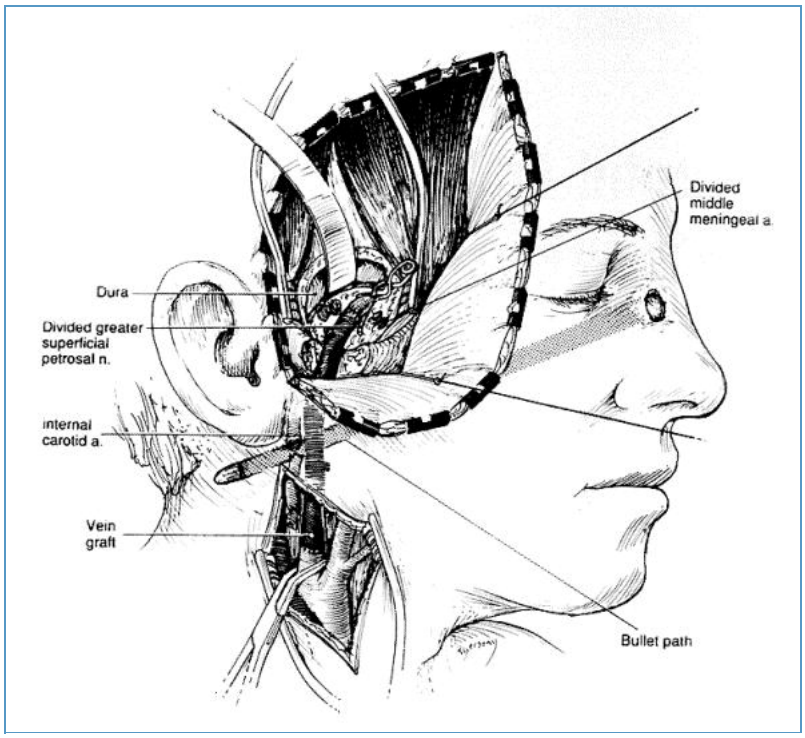

Figure 1. Illustration of case 1, showing the path of injury, anatomic relationships, and surgical exposure for the cervical-to-petrous ICA vein bypass procedure.

CASE STUDIES

Case 1

A 15-year-old girl suffered a GSW to the head, with the entrance at the left midnasal region. The bullet crossed obliquely and caudally to the right, traversing the maxillary sinus, pterygoid plate, and parapharyngeal region, to come to rest in the neck at about the C2 level (see Figure 1). She was intubated in the field and transported to the emergency room, where, on initial evaluation, she was hemodynamically stable with no evidence of other injuries. Her workup included CT scans of the head and neck, cervical and four-vessel cerebral angiogram, TCD, and SPECT

scan. These studies showed a complete occlusion of the proximal cervical right ICA (see Figure 2), with collateral flow through a patent anterior communicating artery, normal head CT scans, normal cerebral perfusion by SPECT scan, and normal flow velocities in the right middle cerebral artery (MCA) with no embolic signatures with 15 minutes of TCD monitoring.

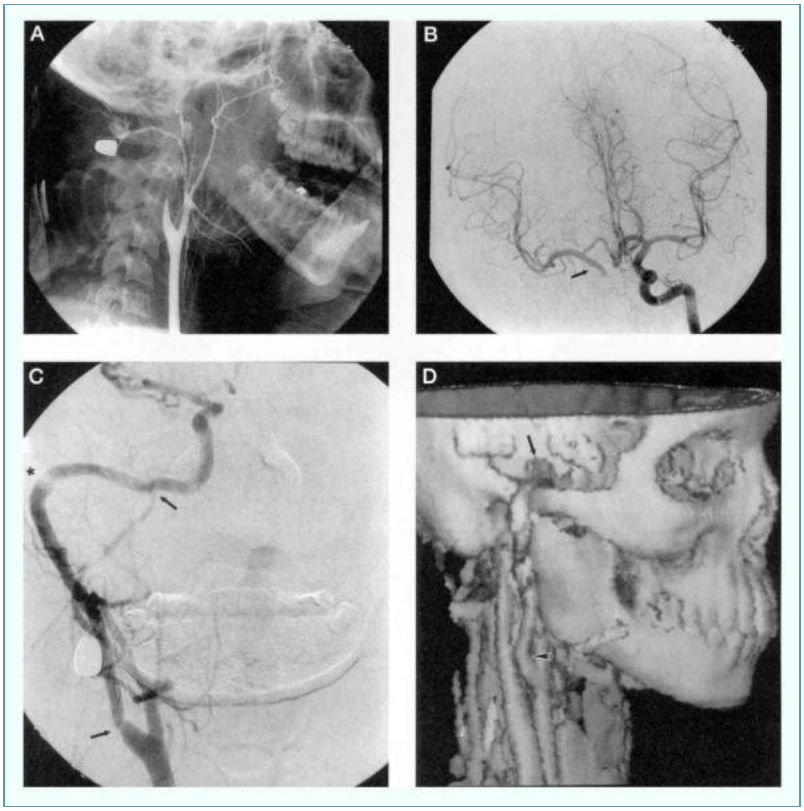

Figure 2. Case 1: (A) A right lateral carotid angiogram demonstrating a complete ICA occlusion. (B) Anteroposterior cerebral angiogram showing collateral circulation from the left ICA to the right ACA and MCA through the circle of Willis. Reflux down the right ICA (arrow) suggests that the petrous portion of the ICA will be patent and suitable for the distal anastomosis. (C) Postoperative angiogram after proximal anastomosis revision showing sites of anastomoses (arrows) and patent graft coursing over the zygoma (*). (D) Postoperative three-dimensional CT angiographic reconstruction showing the site of proximal anastomosis (arrowhead) and small temporal craniotomy (arrow).

She was taken urgently to the operating room, where a reverse saphenous vein was grafted from the proximal cervical to petrous ICA. Postoperative studies (angiography, carotid duplex, and TCD) revealed a patent graft with no emboli. However, the proximal anastomosis was narrowed and the patient was taken back to the operating room for graft revision 1 week later. Follow-up of the revision showed improved caliber of the anastomosis (see Figure 2). Her postoperative course was unremarkable, with no neurologic deficits except a right hypoglossal nerve palsy. Hypoglossal nerve function was not tested preoperatively because of intubation. The path of the bullet and the extent of soft-tissue injury adjacent to the hypoglossal nerve noted intraoperatively makes it likely that this deficit was produced by the initial injury. She has been observed for 3 months, showing no complications from her procedures and with continuing evidence of a patent graft by TCD monitoring.

Case 2

This 34 year-old man suffered a high-velocity GSW from a semiautomatic weapon. The bullet entered at the right medial canthus, traveled laterally and inferiorly to traverse the tip of the left temporal lobe, fracture the petrous bone, and exit from the left mandible. The patient experienced no gross motor deficits at the time of initial evaluation but on subsequent testing was blind and deaf on the left with a left peripheral facial nerve paralysis. Initial workup, including CT scans with fine cuts through the skull base, four-vessel cerebral angiography, and TCD monitoring revealed a left proximal petrous portion ICA pseudoaneurysm adjacent to the middle fossa dura with preserved distal ICA flow (see Figure 3). TCD flow velocities were normal bilaterally in the proximal MCA and anterior cerebral artery (ACA) with no evidence of emboli from the carotid injury.

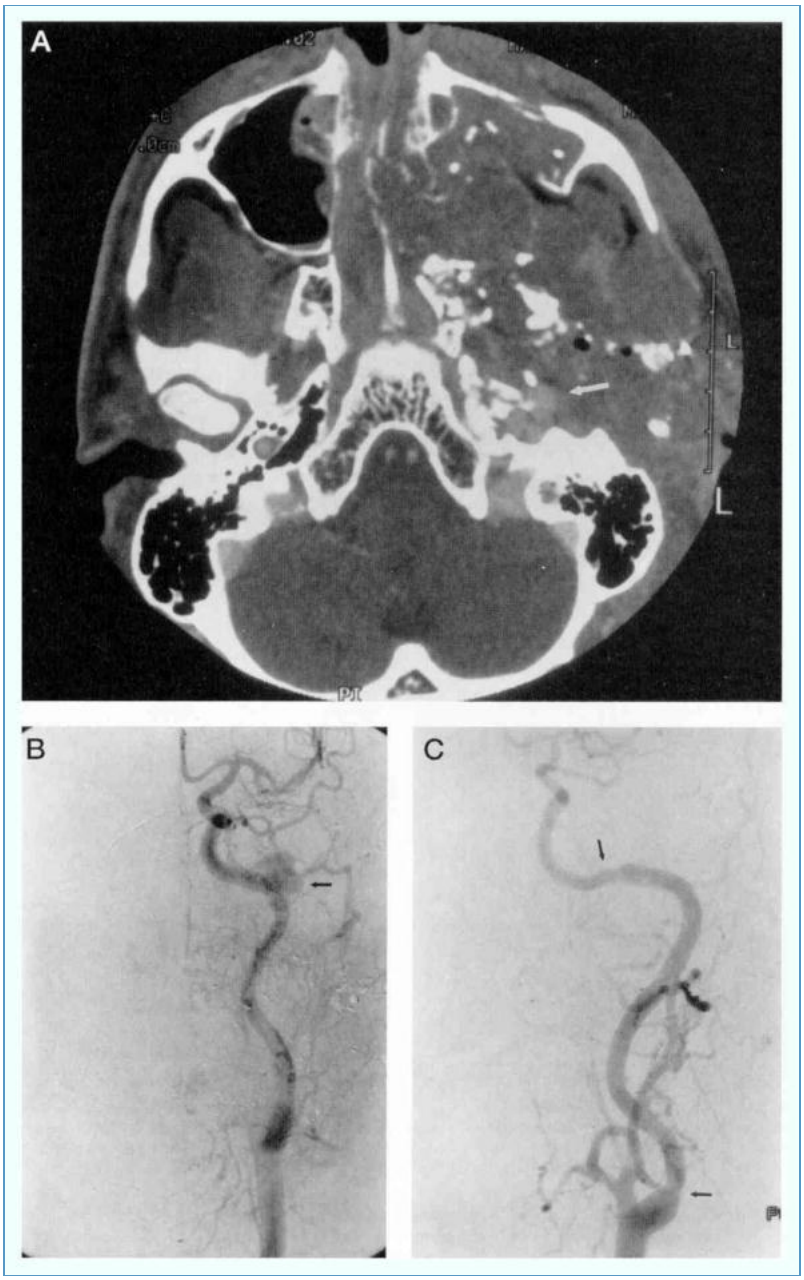

Figure 3. Case 2: (A) Fine-cut skull-base CT scan with constrast showing the lobulated pseudoaneurysm near the entrance to the carotid canal at the skull base (arrow). (B) Preoperative angiogram demonstrating the pseudoaneurysm of the proximal petrous portion of the ICA (arrow). (C) Postoperative angiogram showing patent graft and sites of anastomoses (arrows).

Five weeks after recovery from his brain injury and treatment of his orbital and maxillofacial injuries, a follow-up angiogram revealed enlargement of the pseudoaneurysm. Balloon test occlusion of the right ICA did not elicit neurologic deficits or significant drops in his TCD velocities and he underwent a reverse saphenous vein cervical to petrous carotid bypass grafting procedure without incident. Postoperative angiography revealed a patent graft with no significant stenosis at the anastomoses (see Figure 3). The patient has been observed for 2 years with serial TCD and carotid duplex examinations that confirmed continued graft patency. His neurologic examination remains essentially unchanged, except for return of some facial nerve function.

Case 3

This 22-year-old man suffered a low-velocity GSW to the right side of his face that entered the lip and then traveled through the maxillary sinus, pterygopalatine plate, and middle cranial fossa, with a fragment coming to rest against the right occipital bone. He was transferred intubated from a referral hospital where he was observed to be

moving all extremities. A CT scan showed a small right temporal intracerebral hematoma and angiography revealed an occlusion of the right ICA at the C1 level (see Figure 4). There was some filling of the right supraclinoid carotid artery from the external carotid artery but this did not contribute any blood supply to the right MCA or ACA, which were supplied only by a small posterior communicating artery. In addition, the ACA on the left was atretic and, therefore, both ACAs were filled only through pial collaterals from the left MCA (see Figure 3). The patient underwent emergent intracranial debridement and dural repair, tracheostomy, and arch bar placement. There were no episodes of intraoperative hypotension, but, postoperatively, he was noted to be densely paretic on the left, and subsequent CT and SPECT scans confirmed a massive right MCA distribution infarction. Despite aggressive medical treatment of increased intracranial pressure, the patient eventually herniated and progressed to brain death.

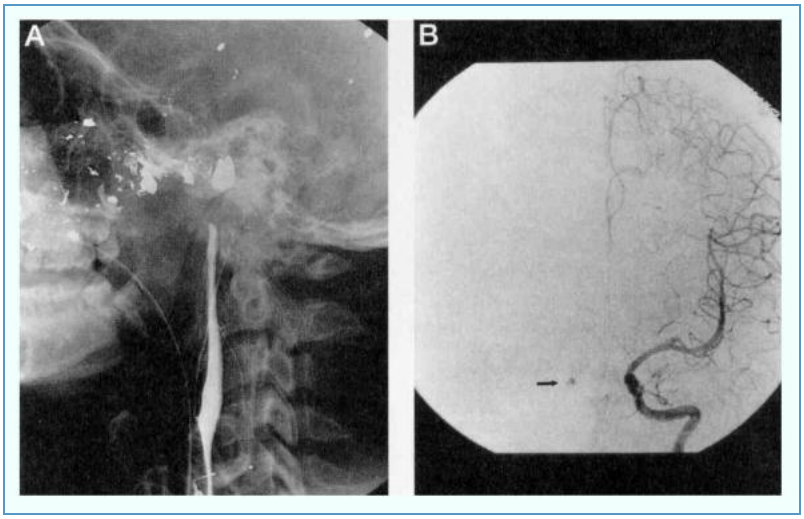

Figure 4. Case 3: (A) Right lateral carotid angiogram showing bullet tract and a high cervical ICA occlusion. (B) Left anteroposterior ICA angiogram showing this patient's unfavorable collateral circulation pattern with an atretic left ACA and no cross-filling through the circle of Willis to the right. The distribution of the left ACA partially fills from pial collaterals supplied by the left MCA and only a blush of contrast material is seen in the right cavernous ICA (arrow).

Case 4

A 24-year-old man suffered a low-velocity GSW that entered at the glabella and traveled obliquely in a path through the right frontal sinus, coming to rest in the right petrous mastoid region. Extensive fractures of the petrous bone, including the carotid canal, were seen on fine-cut CT examinations and four-vessel cerebral angiography revealed a pseudoaneurysm of the right proximal petrous ICA (see Figure 5). On initial clinical evaluation, he displayed extensor posturing with bilaterally fixed and dilated pupils. He underwent emergent craniotomy for debridement of injured brain, dural repair, and cranialization of his frontal sinuses, followed 3 days later by a decompressive craniectomy for intractable elevations of intracranial pressure. Because of a marked neurologic recovery, his pseudoaneurysm was reevaluated 4 weeks after his injury. Repeat angiography revealed an enlarged pseudoaneurysm with patent distal vessels and he was scheduled for a bypass procedure. Two days before his planned bypass, he suffered a massive intracerebral hemorrhage from rupture of his pseudoaneurysm and died (see Figure 5).

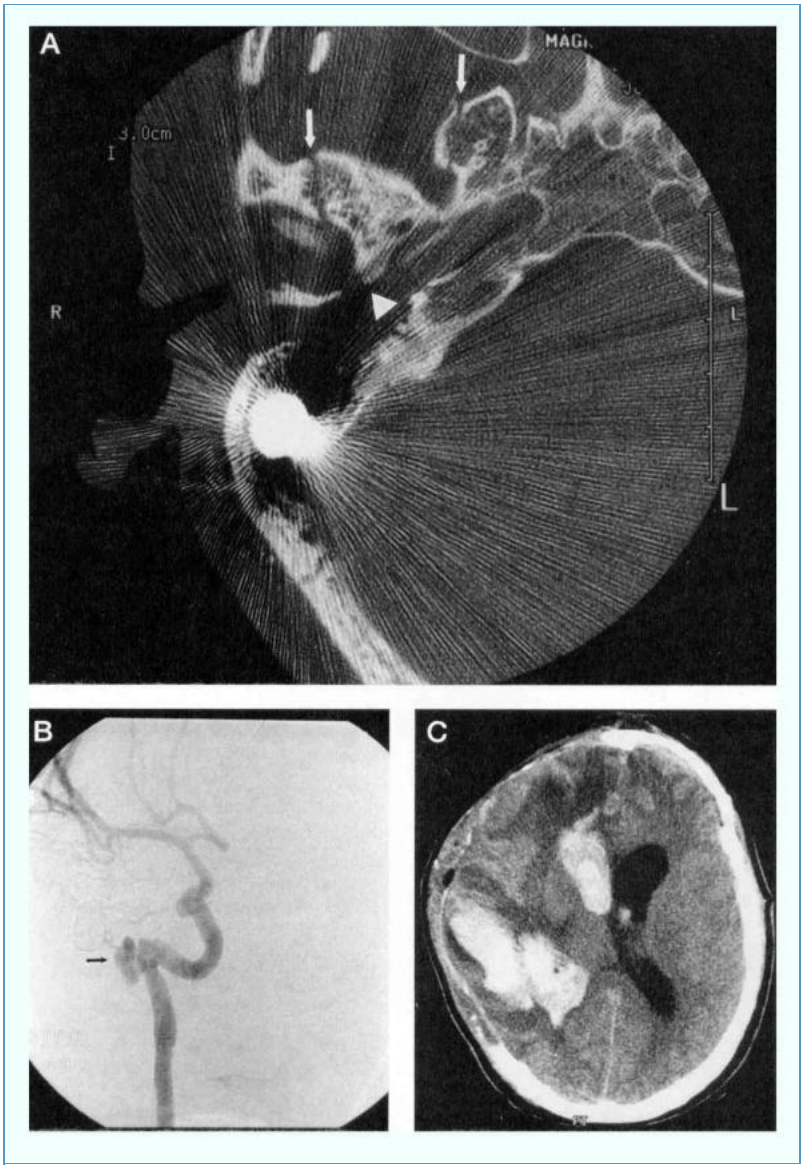

Figure 5. Case 4: (A) Fine-cut temporal bone CT scan showing a bullet fragment lodged in the mastoid bone and fractures (arrows) adjacent to the carotid canal (arrowhead). (B) Oblique right ICA angiogram 4 weeks after admission showing pseudoaneurysm (arrow) that had enlarged compared with study at the time of injury. (C) CT scan showing massive intracerebral hemorrhage from ruptured pseudoaneurysm.

DISCUSSION

Penetrating injuries of the high cervical or skull-base portions of the ICA are relatively rare but are significant because of the unique management challenges they present. Few reports in the literature deal exclusively with penetrating injuries of the high cervical or skull-base portions of the ICA, and larger series of patients with traumatic ICA and common carotid artery injuries often lack sufficient detail or follow-up to judge the longer-term natural history or outcome based on different treatment strategies. These shortcomings are particularly relevant when considering the true incidence of delayed cerebrovascular events after traumatic carotid injury. Thus, it is impossible, based on the available literature, to advocate a dogmatic treatment strategy or algorithm for patients with penetrating high cervical or skull-base ICA injuries. We, therefore, will attempt to briefly highlight the data that bears on the evaluation and treatment of these patients, compare the various surgical approaches to this region, and discuss our cases in this context.

Penetrating Carotid Injury

In a recent population-based survey, traumatic carotid injury accounted for less than 0.2% of hospital admissions for trauma. [5] Of the 82 carotid injuries identified in this study, 55 were caused by penetrating trauma and 27 involved the ICA. Thirty-one of 55 patients with penetrating injuries underwent angiographic examination (16 of the other 24 patients did not undergo angiograms because of shock, and likely sustained major lacerations or avulsions) of the carotid arteries, which revealed pseudoaneurysms in 12 (39%) and occlusion in six (19%). The mortality and stroke rates were much higher in ICA than common carotid artery injury (21 and 41% vs. 11 and 11%, respectively), whereas penetrating trauma had lower stroke rates (15% vs. 56%), but higher mortality (22% vs. 7%) than blunt injuries. The longer term natural history of these lesions is unclear, but in one study of 12 patients with missed carotid injuries, three patients presented with stroke and one with transient ischemic attack. [9] In the series of Sclafani et al., [6] 16 of 46 patients with zone III penetrating neck injuries sustained 18 ICA injuries, including five stenoses (31%), five occlusions (31%), and three aneurysms (19%). Similar arterial lesions have been described in other reports of penetrating skull-base trauma. [2,3]

Traumatic ICA Aneurysms

Traumatic extracranial ICA aneurysms have been reported to rupture, causing epistaxis, otorrhagia, or intracerebral hemorrhage, serve as a nidus for embolic events, or present as cervical or pharyngeal masses often producing cranial neuropathies. [10-17] In 1950, Shumacker and Wayson [18] reported only a 6.6% rate of spontaneous cure of peripheral traumatic aneurysms. In 1990, McCann [15] reviewed the recent literature on carotid arterial aneurysms (13% traumatic) and reported that 40% presented with neurologic symptoms and 56% with a mass. Serial angiographic examinations showed enlargement of all three pseudoaneurysms 1 to 4 weeks in the series of Sclafani et al. [6] In another series of peripheral aneurysms, five of six conservatively managed traumatic pseudoaneurysms enlarged and required surgical intervention. [19] Untreated traumatic intracerebral aneurysms have been reported to carry a mortality rate as high as 50%. [20]

Traumatic Carotid Occlusion

Very little data exist that specifically relate outcome after traumatic carotid injury to the presence of occlusion at presentation. One study of blunt trauma reported a mortality of 40%, and good outcome in only 30%, of 20 patients presenting with a carotid occlusion of whom only one had an attempted surgical repair. [21] Unlike spontaneous ICA dissections, traumatic ICA injuries seem to have a less favorable prognosis when managed conservatively. In a series of 21 ICA dissections after blunt trauma, only 21% of aneurysms and 54% of stenoses showed complete or partial resolution, and 29% of patients were dead or experienced significant deficits at last clinical follow-up. [22] The immediate stroke rate after spontaneous ICA occlusion in patients with atherosclerotic disease has been reported at 25% [23] and 43%, [24] with subsequent risks of stroke of 10% per year (for the first 2 years) and 24% for 5 years, respectively.

The risk of immediate or delayed ischemic complications after intentional carotid occlusion or ligation varies considerably, depending on the patient population and the use of screening tests to judge adequacy of collateral circulation. [25-28] Before the use of test occlusion, stroke rates on abrupt ICA sacrifice ranged from 17 to 45%. [25,27,29] Even after using various paradigms of test occlusion, including clinical testing, cerebral blood flow measurements, and hypotensive challenge, rates of ischemic complications after carotid occlusion/ligation ranged from 3.3 to 22%, [26-28,30-33] with most reporting 3 to 6%, and with higher rates in patients undergoing extensive head and neck procedures for skullbase neoplasms. [28,30,31]

An untreated ICA occlusion or ligated ICA may also develop embolic symptoms from the residual proximal ICA stump [34] or distal "tail." [35] In addition, some reports suggest that, after carotid ligation, patients may be at greater risk of de novo intracranial aneurysm formation and hypertension. [25,36,37] It is assumed that similar risks may apply to patients with an acute traumatic occlusion and are the basis for considering a bypass procedure in young patients with ICA occlusion.

Patient Evaluation

The preoperative assessment of a patient with a suspected skull-base vascular injury is critical for identifying appropriate surgical candidates and to assist in surgical planning. A fine-cut CT scan of the skull base and a routine head CT scan are imperative to establish the presence and extent of injury to the petrous bone and ischemic or hemorrhagic brain injury. A four-vessel cerebral angiogram must also be performed to identify the vascular injury, define collateral circulation patterns, and determine the patency of the distal portion of the petrous ICA.

Other valuable studies that can be used as adjuncts include the SPECT scan, TCD, and carotid duplex examination. SPECT can identify patients with absolute versus relative ischemia, as well as the amount of brain involved before any ischemic changes are evident on CT or MRI scans. [38] These distinctions may help determine which patients may benefit from revascularization without undue risk of hemorrhage into a large ischemic area, even if they present in or progress to coma. TCD provides additional hemodynamic information and is a useful tool for detecting emboli, [39,40] whereas carotid duplex examination helps define the anatomy of the lesion and provides a

noninvasive means to follow graft patency and stenosis postoperatively.

Treatment Options

Depending on the clinical circumstances, a variety of strategies exist for the management of such lesions, including observation, anticoagulation, ligation, balloon occlusion, primary resection and repair, or bypass. [2,3,6-8,14,26,41,42] Although still controversial, most recent series reporting on carotid artery trauma show an improvement in outcome for patients undergoing surgical reconstruction of the injured carotid, particularly in neurologically intact patients. [5,43-45] However, for comatose patients or noncomatose patients with neurologic deficits, surgical reconstruction has shown less consistent benefit. [5,43,44] In his review of the literature on extracranial carotid aneurysms, McCann [15] reported a combined stroke and mortality rate for unoperated aneurysms of 23%, for ligation a rate of 45%, and for carotid reconstruction a rate of 10%. The two patients in this report whose penetrating vascular injuries were managed conservatively died, but good outcomes, and even resolution, of ICA vascular lesions have been described after conservative management, carotid occlusion, or endovascular obliteration of pseudoaneurysms. [1,4,12,46,47]

In the setting of acute penetrating trauma, anticoagulation can be problematic. A stable asymptomatic pseudoaneurysm in a patient not felt to be at risk for intracranial hemorrhage may be observed but the patient should be monitored for embolic phenomena or enlargement. Conservative management of carotid dissection or occlusion is appropriate but the presence of ischemic embolic or hemodynamic-related events warrants consideration for revascularization, particularly when refractory to anticoagulation. Embolic events in the face of carotid occlusion and adequate anticoagulation warrant screening for potential sources from a carotid stump or thrombus "tail" and consideration for intracranial surgical or endovascular balloon trapping of the lesioned segment just proximal to the ophthalmic artery.

Surgical Goals and Approaches

The goals of surgical intervention in patients with acute high cervical occlusion or pseudoaneurysm formation vary from the simple exclusion of a potential embolic or hemorrhagic lesion from the circulation, to the maintenance or reestablishment of normal circulatory patterns to the affected hemisphere. These goals can be met to varying degrees by carotid ligation or vascular bypass procedures, including superficial temporal artery-to-MCA bypass (EC-IC) or a cervical-to-petrous ICA reversed saphenous vein bypass (VBG).

The surgical approach to the high cervical/petrous ICA is challenging because of anatomic constraints that limit direct operative exposure and distal control. The available approaches are designed to either provide direct access or bypass the lesion. Direct ICA repair with vein grafting (if necessary) can be accomplished in some cases by augmenting a standard cervical approach with mobilization of the mandible or sternocleidomastoid and/or digastric muscles. [48-52] These approaches provide limited access to the skull base, and some require manipulation of the facial and lower cranial nerves. Distal control is problematic and can require the blind passage of a Fogarty catheter. A variety of complex "infratemporal" approaches have also been described that provide better access to the petrous ICA and have been used successfully for vein bypass procedures to treat skull-base tumors [53,54] and aneurysms. [14,53,55] However, they require extensive soft-tissue and bony dissection.

Bypass procedures can exclude lesions from the circulation and preserve or reestablish cerebral circulation, but only a few case reports have described their use in traumatic high cervical vascular injuries. [2,3,7,14] Bypasses can be accomplished from the EC-IC vessels, most commonly from the superficial temporal artery to the MCA. [2,3] VBGs, which can be fashioned from the cervical ICA to either the petrous or supraclinoid (intracranial) portion of the ICA, have been used extensively in skull-base tumor surgery and for the treatment of large nontraumatic skull-base intracranial aneurysms. [41,42,54,56-58] Reported long-term graft patency rates for EC-IC saphenous vein grafts are 82 and 73% after 10 and 13 years, respectively. [59]

The cervical-to-petrous VBG has the advantages over an EC-IC bypass of providing immediate restoration of high-volume flow from an extradural approach while simultaneously excluding the underlying lesion from the circulation. In addition, during cross-clamping for the anastomosis in a petrous VBG, there is more potential for collateral circulation. If the petrous ICA is not available to accept a graft, the supraclinoid ICA can be used, [41] but this introduces the morbidity associated with an intracranial approach and limited collateral blood flow while the ICA is cross-clamped to perform the anastomosis. Thus, the cervical-to-petrous ICA VBG has advantages over both the other bypass procedures and the direct approaches by providing better access to the distal ICA and limiting surgical morbidity encountered with infratemporal approaches that require extensive soft-tissue dissection.

In cases of carotid injury with exsanguinating hemorrhage, several potential treatment options exist. One option is balloon occlusion or balloon trapping of an open lesion through an interventional approach if the carotid artery was lacerated and still partially intact. Alternatively, carotid ligation may be performed acutely in an effort to stabilize the patient. If the patient is in a facility where this procedure is not performed, the patient may be transferred to

such a facility after the acute bleeding is controlled. It is likely that reexploration of the injury and carotid to petrous bypass grafting may be performed shortly after an emergency carotid occlusion for bleeding. Reestablishment of flow is likely if the graft procedure is performed soon after ligation.

DISCUSSION OF CASE REPORTS

The following analysis of our cases underscores factors involved in forming the management strategy for a particular patient and suggest where alternative strategies may have been appropriate. The patient in case 1 sustained an asymptomatic carotid occlusion with good hemodynamic reserve, as determined by collateral flow patterns on cerebral angiogram and brain perfusion evaluated with SPECT scan. The decision to operate was based on the patient's young age and the potential long-term morbidity from embolic events, delayed hemodynamic insufficiency, hypertension, and contralateral intracranial aneurysm formation. Alternatively, this patient could have been managed conservatively with or without anticoagulation with careful monitoring of embolic or ischemic events, or by carotid ligation or trapping of the ICA in the cervical region and distally just proximal to ophthalmic artery to eliminate emboli from the carotid stump or distal "tail" of the thrombus.

The patient in case 3 sustained an ICA occlusion similar to that of the patient in case 1, but because of a limited collateral blood supply, he developed a massive and fatal infarction. Initial assessment revealed no focal neurologic deficit and the patient underwent emergent surgery for intracranial wound debridement without revascularization. In retrospect, this patient may have benefited from an emergent bypass. Subsequent CT and SPECT scans were consistent with a hemodynamic basis for his stroke. Although a preoperative SPECT scan would have delayed surgical intervention, it may have altered the operative plan by determining whether the limited collateral flow provided some marginal perfusion or whether there were already areas of brain devoid of blood flow.

The patient in case 2 sustained an enlarging pseudoaneurysm that was successfully managed with a VBG after an extensive workup revealed adequate hemodynamic reserve during temporary occlusion. Although this lesion was asymptomatic, its enlargement and future risk of complications from emboli, rupture, and mass effect prompted surgical intervention. Intervention could have been withheld if serial monitoring revealed no further enlargement of the aneurysm or evidence of emboli. Other options might have included prophylactic or expectant use of anticoagulation, surgical, or balloon carotid occlusion, or endovascular obliteration with preservation of the parent vessel.

The patient in case 4 sustained an enlarging pseudoaneurysm that ruptured, causing a fatal intracerebral hemorrhage just before a planned VBG. Review of contrast-enhanced fine-cut skull-base CT scans suggested a direct connection between the aneurysm and the intracranial compartment through his extensively fractured petrous bone. One anatomic study showed that portions of the carotid canal were covered by dura alone or a thin layer of cartilage in approximately half of the specimens. [60] Theoretically, therefore, any patient with this anatomic configuration may be at risk for direct intracranial extension of a hemorrhage after aneurysm rupture, even in the absence of a fracture.

SUMMARY

Injury to the distal cervical and proximal petrous portion of the ICA is an infrequent, yet potentially catastrophic, sequela of penetrating skull-base injuries. The cases reported here underscore the need for an aggressive approach to the assessment and management of patients with penetrating vascular skull-base injuries. After the initial assessment is completed, any extenuating clinical factors must be factored into an individualized management strategy. Even then, the appropriate treatment choices remain controversial because they have not been rigorously studied and the natural history of a large group of patients with these lesions is not available.

When the goal of treatment is exclusion of the injured portion of the carotid artery and revascularization, a cervical-to-petrous ICA VBG can reduce the potential acute ischemic or delayed embolic or hemorrhagic complications from such injuries and offers the advantages of providing immediate restoration of high-volume flow with good surgical access and minimal risk to intracranial structures. This approach can be accomplished safely in patients with penetrating skull-base trauma and should be considered in selected patients with acute occlusion or pseudoaneurysm of the distal cervical or proximal petrous ICA.

Acknowledgments

The authors thank Paul Schwartz and Janet Schukar for their help in the preparation of the figures, Rick Gersony for the illustration, and Tim Federspiel for his critical review of the manuscript.

REFERENCES

1. Applegate LJ, Pritz MB, Pribram HF: Traumatic pseudoaneurysm of the cervical carotid artery: The value of arteriography. Neurosurgery 26:312, 1990 Ovid Full Text | Bibliographic Links | [Context Link]

2. Fry RE, Fry WJ: Extracranial carotid artery injuries. Surgery 88:581, 1980 Serials Solutions | Bibliographic Links | [Context Link]

3. Gewertz BL, Samson DS, Ditmore QM, et al: Management of penetrating injuries of the internal carotid artery at the base of the skull utilizing extracranial-intracranial bypass. J Trauma 20:365, 1980 Ovid Full Text | Bibliographic Links | [Context Link]

4. Mori S, Feliciani M, Guglielmi G, et al: Regression of an internal carotid artery pseudoaneurysm after therapeutic embolization of a post-traumatic carotid-cavernous fistula secondary to gunshot injury. Neuroradiology 32:226, 1990 [Context Link]

5. Ramadan F, Rutledge R, Oller D, et al: Carotid artery trauma: A review of contemporary trauma center experiences. J Vasc Surg 21:46, 1995 Serials Solutions | Bibliographic Links | [Context Link]

6. Sclafani SJA, Panetta T, Goldstein AS, et al: The management of arterial injuries caused by penetration of zone III of the neck. J Trauma 25:871, 1985 Ovid Full Text | Bibliographic Links | [Context Link]

7. Fitzpatrick BC, Spetzler RF, Ballard JL, et al: Cervical-to-petrous internal carotid artery bypass procedure: Technical note. J Neurosurg 79:138, 1993 Serials Solutions | Bibliographic Links | [Context Link]

8. Newell DW, Grady MS, Nicholls SC: Cervical carotid to petrous carotid bypass for lesions of the upper cervical carotid artery. Ann Vasc Surg 10:76, 1996 Serials Solutions | Bibliographic Links | [Context Link]

9. Perry MO: Complications of missed arterial injuries. J Vasc Surg 17:399, 1993 Serials Solutions | Bibliographic Links | [Context Link]

10. Bhatoe HS, Suryanarayana KV, Gill HS: Recurrent massive epistaxis due to traumatic intracavernous internal carotid artery aneurysm. J Laryngol Otol 109:650, 1995 Serials Solutions | Bibliographic Links | [Context Link]

11. Costantino PD, Russell E, Reisch D, et al: Ruptured petrous carotid aneurysm presenting with otorrhagia and epistaxis. Am J Otol 12:378, 1991 [Context Link]

12. Komiyama M, Yasui T, Nishikawa M: Endovascular treatment of an extracranial internal carotid artery aneurysm at the skull base with mechanically detachable coils: Case report. Neurol Med Chir (Tokyo) 35:745, 1995 Serials Solutions | Bibliographic Links | [Context Link]

13. Lin T: Delayed intracerebral hematoma caused by traumatic intracavernous aneurysm: Case report. Neurosurgery 36:407, 1995 Ovid Full Text | Bibliographic Links | [Context Link]

14. Magnan PE, Branchereau A, Cannoni M: Traumatic aneurysms of the internal carotid artery at the base of the skull: Two cases treated surgically. J Cardiovasc Surg 33:372, 1992 Serials Solutions | Bibliographic Links | [Context Link]

15. McCann RL: Basic data related to peripheral artery aneurysms. Ann Vasc Surg 4:411, 1990 Serials Solutions | Bibliographic Links | [Context Link]

16. Mokri B, Piepgras DG, Sundt TM Jr, et al: Extracranial internal carotid artery aneurysms. Mayo Clin Proc 57:310, 1982 Serials Solutions | Bibliographic Links | [Context Link]

17. Robinson NA, Flotte CT: Traumatic aneurysms of the carotid arteries. Am Surg 40:121, 1972 Serials Solutions | Bibliographic Links | [Context Link]

18. Shumacker HB, Wayson EE: Spontaneous cure of aneurysms and arteriovenous fistulas, with some notes on intrasaccular thrombosis. Am J Surg 79:532, 1950 Serials Solutions | [Context Link]

19. Frykberg ER, Crump JM, Dennis JW, et al: Nonoperative observation of clinically occult arterial injuries: A prospective evaluation. Surgery 109:85, 1991 Serials Solutions | Bibliographic Links | [Context Link]

20. Parkinson D, West M: Traumatic intracranial aneurysms. J Neurosurg 52:11, 1980 Serials Solutions | Bibliographic Links | [Context Link]

21. Cogbill TH, Moore EE, Meissner M, et al: The spectrum of blunt injury to the carotid artery: A multicenter perspective. J Trauma 37:473, 1994 Ovid Full Text | Bibliographic Links | [Context Link]

22. Mokri B: Traumatic and spontaneous extracranial internal carotid artery dissections. J Neurol 237:356, 1990 Serials Solutions | Bibliographic Links | [Context Link]

23. Nicholls SC, Bergelin R, Strandness DE: Neurologic sequelae of unilateral carotid occlusion: Immediate and late. J Vasc Surg 10:542, 1989 Serials Solutions | Bibliographic Links | [Context Link]

24. Faught WE, van Bemmelen PS, Mattos MA, et al: Presentation and natural history of internal carotid artery occlusion. J Vasc Surg 18:512, 1993 Serials Solutions | Bibliographic Links | [Context Link]

25. Jha AN, Butler P, Lye R, et al: Carotid ligation: What happens in the long term. J Neurol Neurosurg Psychiatry 49:893, 1986 Serials Solutions | Bibliographic Links | [Context Link]

26. Larson JJ, Tew JM Jr, Tomsick TA, et al: Treatment of aneurysms of the internal carotid artery by intravascular balloon occlusion: Long-term follow-up of 58 patients. Neurosurgery 36:26, 1995 Serials Solutions | Bibliographic Links | [Context Link]

27. Linskey ME, Jungreis CA, Yonas H, et al: Stroke risk after abrupt internal carotid artery sacrifice: Accuracy of preoperative assessment with balloon test occlusion and stable xenon-enhanced CT. AJNR Am J Neuroradiol 5:829, 1994 [Context Link]

28. Origitano TC, al-Mefty O, Leonetti JP, et al: Vascular considerations and complications in cranial base surgery. Neurosurgery 35:351, 1994 [Context Link]

29. Moore O, Baker HW: Carotid-artery ligation in surgery of the head and neck. Cancer 8:712, 1955 Serials Solutions | Bibliographic Links | [Context Link]

30. McIvor NP, Willinsky RA, TerBrugge KG, et al: Validity of test occlusion studies prior to internal carotid artery sacrifice. Head Neck 16:11, 1994 Serials Solutions | Bibliographic Links | [Context Link]

31. Sekhar LN, Patel SJ: Permanent occlusion of the internal carotid artery during skull-base and vascular surgery: Is it really safe? Am J Otol 14:421, 1993 Ovid Full Text | Bibliographic Links | [Context Link]

32. Standard SC, Ahuja A, Guterman LR, et al: Balloon test occlusion of the internal carotid artery with hypotensive challenge. AJNR Am J Neuroradiol 16:1453, 1995 Serials Solutions | Bibliographic Links | [Context Link]

33. Zane RS, Aeschbacher P, Moll C, et al: Carotid occlusion without reconstruction: A safe surgical option in selected patients. Am J Otol 16:353, 1995 Ovid Full Text | Bibliographic Links | [Context Link]

34. Barnett HJM, Peerless SJ, Kaufman JCE: "Stump" of internal carotid artery: A source for further cerebral embolic ischemia. Stroke 9:448, 1978 Ovid Full Text | Bibliographic Links | [Context Link]

35. Finklestein S, Kleinman GM, Cuneo R, et al: Delayed stroke following carotid occlusion. Neurology 30:84, 1980 Serials Solutions | Bibliographic Links | [Context Link]

36. Dyste GN, Beck DW: De novo aneurysm formation following carotid ligation: Case report and review of the literature. Neurosurgery 24:88, 1989 Ovid Full Text | Bibliographic Links | [Context Link]

37. Winn HR, Richardson AE, Jane JA: Late morbidity and mortality of common carotid ligation for posterior communicating artery aneurysms: A comparison to conservative treatment. J Neurosurg 47:727, 1977 Serials Solutions | Bibliographic Links | [Context Link]

38. Masdeu JC, Brass LM: SPECT imaging of stroke. J Neuroimaging 5(Suppl):S14, 1995 Serials Solutions | Bibliographic Links | [Context Link]

39. Newell DW: Transcranial Doppler ultrasonography. Neurosurg Clin North Am 5:619, 1994 Serials Solutions | Bibliographic Links | [Context Link]

40. Spencer MP, Thomas GI, Nicholls SC, et al: Detection of middle cerebral artery emboli during carotid endarterectomy using transcranial Doppler ultrasonography. Stroke 21:415, 1990 Ovid Full Text | Bibliographic Links | [Context Link]

41. Morgan MK, Sekhon LHS: Extracranial-intracranial saphenous vein bypass for carotid or vertebral artery dissections: A report of six cases. J Neurosurg 80:237, 1994 Serials Solutions | Bibliographic Links | [Context Link]

42. Schievink WI, Piepgras DG, McCaffrey TV, et al: Surgical treatment of extracranial internal carotid artery dissecting aneurysms. Neurosurgery 35:809, 1994 Ovid Full Text | Bibliographic Links | [Context Link]

43. Liekweg WG, Greenfield LJ: Management of penetrating carotid arterial injury. Ann Surg 188:587, 1978 Ovid Full Text | Bibliographic Links | [Context Link]

44. Timberlake GA, Rice JC, Kerstein MD, et al: Penetrating injury to the carotid artery: A reappraisal of management. Am Surg 55:154, 1989 Serials Solutions | Bibliographic Links | [Context Link]

45. Weaver FA, Yellin AE, Wagner WH, et al: The role of arterial reconstruction in penetrating carotid injuries. Arch Surg 123:1106, 1988 Serials Solutions | Bibliographic Links | [Context Link]

46. Halbach VV, Higashida RT, Hieshima GB, et al: Aneurysms of the petrous portion of the internal carotid artery: Results of treatment with endovascular or surgical occlusion. AJNR Am J Neuroradiol 11:253, 1990 Serials Solutions | Bibliographic Links | [Context Link]

47. Kinugasa K, Mandai S, Tsuchida S, et al: Direct thrombosis of a pseudoaneurysm after obliteration of a carotid-cavernous fistula with cellulose acetate polymer: Technical case report. Neurosurgery 35:755, 1994 [Context Link]

48. Batzdorf U, Gregorius FK: Surgical exposure of the high cervical carotid artery: Experimental study and review. Neurosurgery 13:657, 1983 Ovid Full Text | Bibliographic Links | [Context Link]

49. Mock CN, Lilly MP, McRae RG, et al: Selection of the approach to the distal internal carotid artery from the second cervical vertebra to the base of the skull. J Vasc Surg 13:846, 1991 Serials Solutions | Bibliographic Links | [Context Link]

50. Nelson SR, Schow SR, Stein SM, et al: Enhanced surgical exposure for the high extracranial internal carotid artery. Ann Vasc Surg 6:467, 1992 Serials Solutions | Bibliographic Links | [Context Link]

51. Purdue GF, Pellegrini RV, Arena SA: Aneurysms of the high internal carotid artery: A new approach. Surgery 89:268, 1981 Serials Solutions | Bibliographic Links | [Context Link]

52. Shaha A, Phillips T, Scalea T, et al: Exposure of the internal carotid artery near the skull base: The posterolateral anatomic approach. J Vasc Surg 8:618, 1988 Serials Solutions | Bibliographic Links | [Context Link]

53. Fisch U, Oldring DJ, Senning A: Surgical therapy of internal carotid artery lesion of the skull base and temporal bone. Otolaryngol Head Neck Surg 88:548, 1980 [Context Link]

54. Sekhar LN, Schramm VL, Jones NF, et al: Operative exposure and management of the petrous and upper cervical internal carotid artery. Neurosurgery 19:967, 1986 Ovid Full Text | Bibliographic Links | [Context Link]

55. Glasscock ME III, Smith PG, Bond AG, et al: Management of aneurysms of the petrous portion of the internal carotid artery by resection and primary anastomosis. Laryngoscope 93:1445, 1983 [Context Link]

56. Sen C, Sekhar LN: Direct vein graft reconstruction of the cavernous, petrous, and upper cervical internal carotid artery: Lessons learned from 30 cases. Neurosurgery 30:732, 1992 Ovid Full Text | Bibliographic Links | [Context Link]

57. Sundt TM, Pearson BW, Piepgras DG, et al: Surgical management of aneurysms of the distal extracranial internal carotid artery. J Neurosurg 64:169, 1986 Serials Solutions | Bibliographic Links | [Context Link]

58. Sundt TM, Piepgras DG, Marsh WR, et al: Saphenous vein bypass grafts for giant aneurysms and intracranial occlusive disease. J Neurosurg 65:439, 1986 Serials Solutions | Bibliographic Links | [Context Link]

59. Regli L, Piepgras DG, Hansen KK: Late patency of long saphenous vein bypass grafts to the anterior and posterior cerebral circulation. J Neurosurg 83:806, 1995 Serials Solutions | Bibliographic Links | [Context Link]

60. Paullus WS, Pait TG, Rhoton AL Jr: Microsurgical exposure of the petrous portion of the carotid artery. J Neurosurg 47:713, 1977 Serials Solutions | Bibliographic Links | [Context Link]

IMAGE GALLERY

Select All Export Selected to PowerPoint

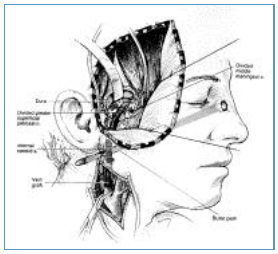

Figure 1

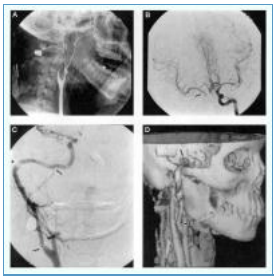

Figure 2

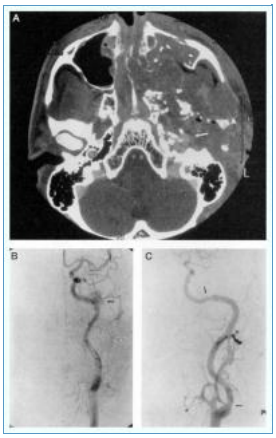

Figure 3

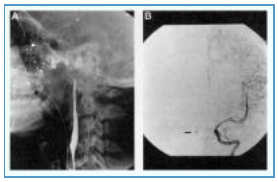

Figure 4

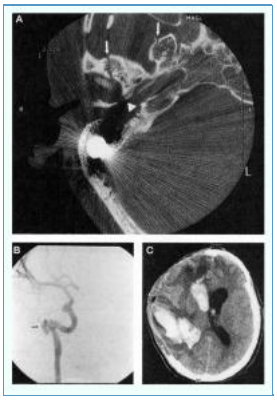

Figure 5

Back to Top