Carotid Endarterectomy

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Checklist: Carotid Endarterectomy (Insertion of Indwelling Shunt)

Equipment needed	Procedural steps
OR technicians • Soft-shoe Fogarty clamp (for common carotid) • Two (2) bulldog clamps (for ECA and ICA) • Indwelling shunt (centerpoint pre-marked) Nursing • Two (2) pre-selected shunts are available in the room (one opened on tray) Anesthesia/Neuromonitoring • Establish neurological baseline with EEG and SSEP • Appropriate anesthesia to ensure insure accurate readings • Availability of additional resources (neurologist) if additional interpretation is required • Pentobarbital/propofol available Neurosurgery • Confirms all equipment present and functional • Discuss with team criteria for shunting	Identify and dissect • Common, internal, and external carotid artery (gently) • Superior thyroid artery • Place sutures around each Initiate and engage • 5000 Units heparin IV • Pott's tie of the superior thyroid artery • Notify anesthesia and team of imminent cross-clamping • Cross clamp (ICA, CCA, ECA in that order!) • Identify criteria for shunting based on EEG, SSEP, and blood pressure. If necessary: • Raise BP 15% (as tolerated) • Complete arteriotomy with Pott's scissors • Place distal (ICA) part of shunt into ICA and look for back-bleeding • Clamp shunt and place proximal portion into CCA as you loosen the ligature on the CCA • Secure with Rommel • Assess EEG/SSEP • Proceed with endarterectomy Removal of shunt • Primary or patch closure of endarterectomy • Prior to final stitches, clamp shunt in two regions proximal and distal to midpoint of shunt • Cut shunt and remove common carotid side, back-bleed, and reclamp • Remove shunt from ICA side, back-bleed, and reclamp • Close remaining portion of patch or arteriotomy

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Complication Avoidance Flowchart

Complication	Cause	Remedy	Avoidance
Stroke	Inadequate perfusion at cross-clamping	Increase BP Shunt placement	Be prepared to shunt
Stroke	Embolus at surgery	Too late	Expose ICA well above plaque Give heparin bolus early Gentle dissection of vessels Place distal shunt into normal vessel, not plaque
Nerve injury	Traction or transection	Recovery with time	No fixed retractors Preserve all nerves Strong knowledge of anatomy
Wound infection (rare)	Comorbidities Diabetes	Wound management, drainage, antimicrobial therapy	Bloodless dry surgery Use surgical drain in sheath Copious irrigation
MI	Systemic vascular disease	Medical therapy in ICU Give aspirin every day including day of surgery	Pre-op cardiac evaluation Skilled anesthesia Some consider locoregional anesthesia

Introduction

In the hands of experienced surgeons, anesthesiologists, and neurocritical care intensivists, patients undergoing CEA experience a low incidence of complications, with common complications tending to be transient [1–8]. But despite the rare incidence of devastating complications, their clinical course can evolve rapidly, and patients may deteriorate unless there is immediate recognition of untoward events and quick intervention to reverse them.

Procedural Overview

Our Technique for CEA

In planning for successful CEA, the surgeon has technique choices to make, such as use of local or general anesthesia, intraprocedural neuroprotective agents, intraoperative monitoring and the linked need for intraoperative carotid shunting, and endarterectomy type, whether linear or eversion, primary repair, microscopic repair, or carotid patch grafting.

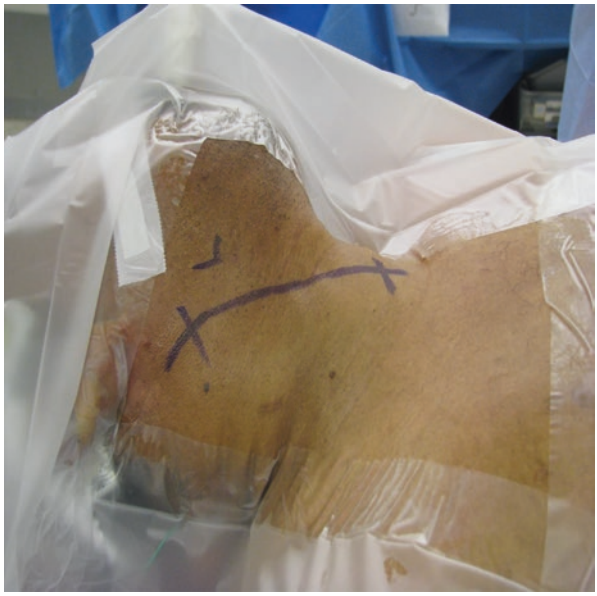

Fig. 12.1 Standard vertical incision for a right CEA. The incision is along the anterior sternocleidomastoid edge, and the angle of the mandible is marked for height orientation. The head is turned away from the side of interest to facilitate exposure of the ICA

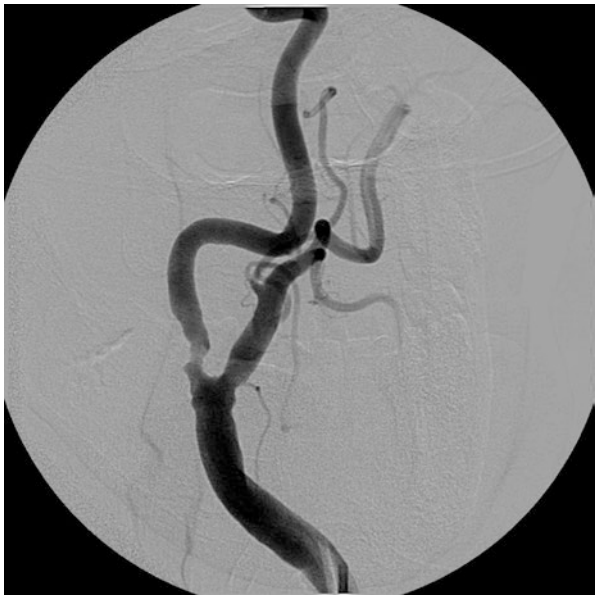

Fig. 12.2 Antero-posterior DSA image of a medially rotated internal carotid configuration, which will make surgical exposure and potential shunt insertion more difficult than in a routine case. It is important to understand this anatomy in the preoperative analysis of the case

As I perform and teach carotid surgery, the patient is positioned supine with the head turned variably to the contralateral side (Fig. 12.1), according to the ICA and ECA relationship on preprocedural imaging (Fig. 12.2), and with the neck slightly extended by placement of an interscapular roll. I utilize concomitant EEG and SSEP monitoring. A linear so assess ischemia and the need for indwelling shunt. The

skin incision is vertical, made along the anterior border of the sternocleidomastoid muscle. The platysma is then incised sharply, and blunt self-retaining retractors are placed, staying superficial on the medial side to prevent injury to the laryngeal nerves. (Once deep to the platysma layer, we use only blunt fishhook-type (Lone Star) retraction, to prevent nerve injury). The dissection is then carried along the anterior aspect of the sternocleidomastoid muscle. The carotid sheath is entered, and the internal jugular vein and ansa cervicalis nerve are identified and mobilized laterally and medially, respectively. The common facial vein is then found medially and ligated to further facilitate lateral mobilization of the internal jugular.

The surgeon should carefully dissect the common carotid artery (CCA) while avoiding injury to the vagus nerve, which typically lies along the posterior aspect of the carotid vessels. Then the dissection is carried rostrally to expose the origins of the internal carotid artery (ICA), external carotid artery (ECA), superior thyroid artery, and occasionally some anomalous ECA branches. Anesthesia should be aware at this time of possible reflex bradycardia and hypotension that may occur with stimulation of the carotid bulb, although with increasing experience we almost never see this and essentially never need to anesthetize the carotid bulb. As the dissection continues rostrally, the hypoglossal nerve should be identified and should be carefully mobilized medially. Once the ICA distal to the plaque has been exposed, 0 silk ties are passed around the CCA, ICA, and ECA, with a rubber Rummel tourniquet to the CCA, in preparation for possible shunting. The superior thyroid artery is secured with a temporary "Pott's tie" 0 silk ligature. At the first visualization of the CCA, before the real arterial dissection begins, the patient is given an intravenous dose of 5000 IU heparin, so that intravascular thrombosis does not occur during the period of temporary carotid occlusion. (We do not routinely re-dose heparin, we do not routinely check ACT levels, and we never use protamine for heparin reversal.) Atraumatic vascular clamps are then applied as follows: first, on the ICA, then on the CCA, and, finally, on the ECA. The superior thyroid artery is occluded with the encircling Pott's tie. *Note that the ICA is occluded first and never declamped until the repair is complete (unless a shunt is placed)*. This sequence affords maximal brain protection from embolized plaque or thrombus. An arteriotomy is then begun with an #11 blade in the CCA just below the bifurcation and is extended into the internal carotid artery distal to the plaque using Potts scissors (Fig. 12.2). The proximal end of the plaque is circumferentially separated from the wall of the artery with the use of a Penfield 4 dissector, which allows complete transection of the plaque proximally. The plaque is then elevated and carefully separated from the wall of the artery at the origin of the ECA through a wall inversion technique, and the stump is sharply divided. This is followed by a similar but more delicate feathering technique, for the ICA plaque to prevent flap formation.

During cross-clamping, I place a shunt when the monitoring neurologist identifies a change in the EEG or SSEP or both after cross-clamping. Our threshold is low; we shunt immediately and without hesitation for any perceived monitoring change. Additionally, in our experience, the arteriotomy is closed with a patch graft, which has reduced to 0 our rates of acute post-op occlusion and long-term restenosis. This is sutured with two limbs, first from distal to proximal on the medial side with the use of a running 6-0 Prolene suture and then two suture lines on the lateral side that meet in the middle and are tied together at their meeting point after

backbleeding and evacuation of all air and debris from the lumen. After the final knot is secured, we complete the declamping first by unclamping the ECA, then the CCA, waiting 10 s, and, finally, opening the ICA. In this way any potential residual air or debris passes harmlessly into the ECA circulation and not to the brain.

Complication Avoidance and Management

Complications associated with CEA can be *wound related*, *systemic*, or *neurological*. Wound-related complications include hoarseness, nerve injury, hematoma, arterial disintegrity, and infection. Systemic complications are primarily cardiac, including MI. Neurological complications include TIA and stroke.

Arterial leak or disruption is, fortunately, an exceedingly rare complication of CEA. (We have actually never experienced an acute arterial leak, nor have we ever had to reexplore a fresh CEA patient for hematoma or airway compromise, and we attribute this good fortune to meticulous hemostasis and assiduous attention to the details of the arterial closure.) If there is an acute arterial leak, patients will have a swollen neck and will demonstrate signs of airway compromise such as dyspnea and dysphagia; they may also demonstrate symptoms of cerebral ischemia. Prompt protection of the airway is imperative as an enlarging hematoma can cause tracheal deviation, making intubation progressively more difficult. After securing an airway, the correction can only be achieved by reoperation. In desperate cases, the incision may be opened at the bedside to alleviate pressure from the hematoma (I have actually never done this). If the wound can be explored at that time, theoretically the surgeon can locate the artery and clamp it to maintain temporary hemostasis and allow time to get the patient to the operating room. Obviously this is a salvage situation that anyone would wish to avoid if at all possible. In a delayed loss of arterial integrity, patients may present days to weeks after a CEA, though the pattern of symptoms progresses without the same rapidity. A mass below the skin may slowly form and be pulsatile. Imaging should be obtained to confirm the diagnosis, and carotid duplex or angiography can identify a pseudoaneurysm at the arteriotomy site. Fevers and cellulitis often present at this time as the etiology of pseudoaneurysms is often linked to postoperative wound infections. The Texas Heart Institute's series of 4991 CEAs found that 35% of postoperative pseudoaneurysms could be directly linked to *Staphylococcus* or *streptococci* wound infections. Other patients in their series with pseudoaneurysms, but no frank evidence of wound infections, did have prolonged operative times, postoperative hypertension, and bacteremia—all factors that commonly contribute to wound infections [9]. Given their infectious etiology, treatment for these patients should include debridement of the surgical wound and washout of any potentially infectious nidus. The arteriotomy site should be examined, and direct repair should be carried out if possible; otherwise, other vascular strategies, like bypass of the diseased segment, may be needed. Realistically, in the present era, we would look first at an endovascular strategy as the low-risk option for arterial disruption, with or without infection, when the presentation is delayed.

Cerebral ischemia that follows CEA may or may not be symptomatic, but has been reported in approximately 5% of CEA patients. Neurological symptoms in the immediate postoperative period must always be evaluated promptly with imaging of

the arterial repair. Minor episodes, like TIAs, or major stroke, like hemiparesis, may reflect embolic phenomena from a denuded arteriotomy bed or may signal a complete occlusion of the carotid. In the rare case where a patient shows neurological changes post-op, we evaluate the repair immediately with the fastest available method, usually CTA, or, if not, duplex ultrasound. Most cases prove to be widely patent repairs and will resolve spontaneously. For this reason, we do not rush back to surgery without imaging; it is almost never necessary and for the most part the problem resolves on its own. If the vessel is occluded, of course, an immediate exploration and reconstruction is the best option. In our practice acute occlusion has been essentially eliminated by the use of universal Hemashield patch grafting. This has been confirmed by others. In a series by Sundt et al., patients treated with patch grafts had a 0.8% rate of occlusion versus a 4% rate among patients treated with primary closure, an improved outcome in patients with patch closure (Figs. 12.3 and 12.4).

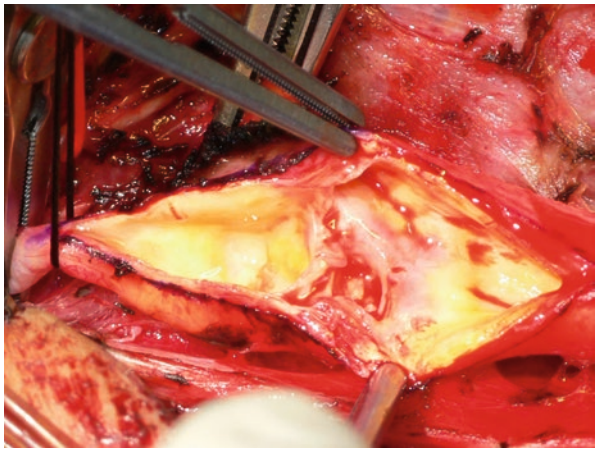

Fig. 12.3 Surgical exposure of an open carotid bifurcation showing a complex ulcerated plaque and an atherosclerotic web, both contributing o carotid stenosis. The plaque removal has not yet started

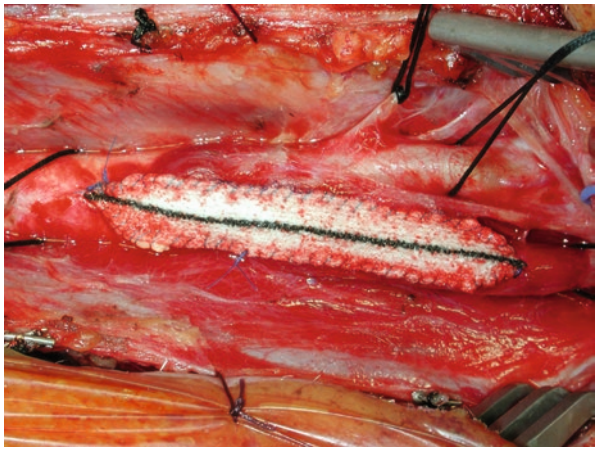

Fig. 12.4 The competed arterial repair with a Hemashield patch angioplasty, following unclamping, in a left carotid case

A severe cerebral hyperperfusion state may occur in <1–3% of CEA patients. The exact etiology is unknown, but chronic ischemia may disrupt the normal autoregulation of cerebral arteries, which may not be able to handle the correction of cerebral blood flow. Several studies have evaluated postoperative cerebral perfusion and found transient elevations in cerebral blood flow of 20–40% for several days following CEA. In a hyperperfusion state caused by flow dysregulation, however, cerebral blood flow may increase 100–200% above baseline. Typically, these pathologic elevations begin 3–4 days following a CEA, but they may even occur up to 1 month from the time of surgery. Patients suffering from hyperperfusion syndrome may show signs of headache, ipsilateral eye pain, face pain, vomiting, confusion, visual disturbances, focal motor seizures, or focal neurologic deficits. If hyperperfusion syndrome is suspected, immediate steps should be taken to lower blood pressure to a normal range, usually with labetalol or clonidine. Antihypertensive therapy with strict normotensive goals is necessary for at least 6 months, while cerebral autoregulation is reestablished. A CT scan has been recommended as the first-line imaging study of choice and may demonstrate ipsilateral hemispheric petechial hemorrhages, ipsilateral basal ganglia hemorrhage, and parieto-occipital white mater edema, all of which may be indicative of hyperperfusion syndrome.

While acute hemorrhagic and ischemic complications are worrisome, the more common issue seen is transient cranial nerve injury, which occurs with low but predictable regularity following CEA [10]. In the NASCET trial, cranial nerve injury was found to occur in 8.6% of patients. Studies carried out more recently have demonstrated lower (~5%) rates. Most nerve deficits will result from traction injury of the hypoglossal nerve or the marginal mandibular branch of the facial nerve. Also at risk in the standard exposure, however, are the recurrent laryngeal, vagus, and (occasionally) the accessory nerves, as well as the cervical ganglia of the sympathetic trunk.

Preoperative nerve evaluation is important, particularly in the case of hypoglossal or recurrent laryngeal nerve palsies contralateral to the proposed CEA. We have seen residual nerve injuries from contralateral carotid surgeries, as well as tracheostomy and other neck procedures. When there are contralateral nerve palsies, we prefer an endovascular strategy to avoid the devastating possibility of a bilateral laryngeal or hypoglossal injury.

Postoperative cranial nerve palsy incidence is best predicted by the length and anatomical difficulty of the CEA [10]. Surgeries lasting greater than 2 h have a 50% greater chance of causing a cranial nerve injury for each additional 30 min of operative time. In our practice, as mentioned earlier, we have moved away from fixed retractors to a fishhook Lone Star retraction system, and this change has markedly reduced our incidence of nerve injury to almost 0.

Durability and Rate of Recurrence

Recurrent carotid stenosis after carotid endarterectomy can occur as a result of neointimal hyperplasia (for stenosis recurring within 24 months of surgery) and from recurrent atherosclerosis (after 24 months). In the carotid artery, the bifurcation

creates shear stress related to turbulent, high pressure arterial blood flow leading to endothelial damage and a focal, recurrent inflammatory cascade that leads to progressive deposition of atheromatous plaque which gradually compromises the integrity of the carotid arterial lumen. These forces remain present after CEA, making the patient susceptible to recurrent atherosclerosis over the course of several years.

A small but finite incidence of recurrent carotid stenosis occurs after CEA. Most studies quote a symptomatic recurrence rate of approximately 4–5%. In one study of noninvasive follow-up after CEA, a 4.8% recurrence rate of symptomatic carotid restenosis was found, with an additional 6.6% asymptomatic restenosis. As mentioned earlier, authors who use universal patch graft techniques have reported lower restenosis numbers (1% symptomatic, 4–5% total at 2 years follow-up).

Aside from technical shortfalls at the time of surgery, it is difficult to establish risk factors associated with recurrent carotid stenosis. Continuation of tobacco smoking postoperatively has been accepted as a significant risk factor, although hypertension, diabetes mellitus, family history, aspirin use, coronary artery disease, and lipid studies were not found to be significant risk factors.

Redo CEA can be performed for restenosis, although, in general, it should be regarded as a high-risk procedure, as reoperation can lead to more cranial nerve injuries and local complications. There has also been an increased incidence of stroke reported in patients undergoing redo CEA. Accordingly, and contrary to our previous teachings, we now recommend CAS for recurrent *symptomatic* carotid disease and a course of watchful waiting with annual imaging for *asymptomatic* recurrence [11, 12]. In truth, with the adoption of universal patch grafting in our practice for a number of years, we see almost no recurrent disease in our surgical patients, and most of the patients whom we either follow or refer for CAS have been referred for complex management after surgery was done elsewhere.

Clinical and Radiographic Follow-Up

How should routine post-CEA patients be followed long term? Given the quick availability and noninvasive nature of Doppler ultrasonography, we elect to perform an ultrasound on the patient the day after CEA to verify wide patency and establish a baseline. We follow our CEA patients for life and typically obtain a repeat Doppler ultrasound at 3 months and then annually. Once stability has been established over an extended period, surveillance at longer intervals may be appropriate. Termination of surveillance is reasonable when the patient is no longer a candidate for intervention.

Summary of Complications

Potential Complications that are of concern:

1. Stroke
2. Nerve injury
3. Arterial problems, leaks, aneurysms, wound infections
4. Medical issues, primarily MI

1. Stroke: Why would someone have a stroke? (A) There isn't enough blood flow to the brain. The remedy for this involves neurological monitoring, in our case EEG/SSEP, and placement of an indwelling shunt if the monitoring changes indicating cerebral ischemia. CEA complications can be *acute* or *delayed*. (B) An embolus of plaque, clot, or air passes up the ICA to the brain. The remedy for this is meticulous technique, giving adequate heparin, dissecting with a low-touch technique, and never clamping across a plaque or passing a shunt through an area of plaque. The ICA must be protected at all times and never declamped until the repair is complete, declamping at other times could expose the ICA and the brain to emboli. Finally, the sequence of declamping must be studied and memorized so that any debris is flushed into the ECA, never the ICA. (C) The vessel occludes post-op. The remedy for this is meticulous technique and, in our practice, universal patch grating.
2. Nerve injury: The surgeon must be familiar with the location and function of the hypoglossal, vagus, accessory, recurrent and superior laryngeal nerves, and the ansa hypoglossi. Injuries can occur from traction, transection, or cautery. The remedy is, first, the use of fishhook retractors rather than fixed retractors and, second, adopting a policy as we have of never cutting any nerves (like the ansa) instead choosing to work in and around them.
3. Arterial problems, hematomas, leaks, aneurysms, wound infections: Fortunately these are almost never events. Wound infections are almost nonexistent, I have seen only one in 30 years, which resolved with exploration and antibiotic treatment. I have seen one delayed (4 years) asymptomatic false aneurysm, which we treated with a stent. We take every precaution to prevent leaks, and we do not close unless the wound is arid dry. We place a medium hemovac in the carotid sheath in every case. Regarding the suture line, we use reinforcing sutures routinely to prevent unraveling (which I have never seen happen), and we never grasp the Prolene suture with forceps, which would weaken it. If someone mistakenly grasps it, I oversew that entire wall again for certainty with a new suture. If there is a hematoma post-op, as can happen in prosthetic cardiac valve patients who need heparin maintained post-op, one must assure the integrity of the suture line immediately. We used to do angiography, but now we do CTA or MRA to acquire this data. *I have never in my career reoperated a carotid surgery for a wound hematoma*. I attribute this to careful and meticulous technique at the primary operation.
4. Medical issues, primarily MI: The remedy for these issues is a proper preoperative medical clearance, a skilled anesthetist, patients housed in the NSICU post-op, and continuation of daily aspirin before, during, and after the operation. The phenomenon of dysautoregulation hemorrhage in patients with tight stenosis is avoided by ICU management with tight BP control.

Conclusion

Carotid endarterectomy must have a very low complication rate in order to benefit patients, especially in the asymptomatic population. Thus, it is imperative that in addition to proper patient selection and an appropriate medical work-up,

meticulous care be taken at each step of the procedure to ensure success. Anticipating and understanding the critical elements that can cause a complication will help to ensure success.

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