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CHAPTER 1.4

Carotid Endarterectomy

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Carotid Endarterectomy

Surgical Considerations

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Description: Carotid endarterectomy (CEA) is frequently used to treat severe atherosclerotic occlusive disease involving internal carotid arteries at the common carotid artery bifurcation. Atherosclerotic carotid artery disease commonly causes thromboembolic or hemodynamic stroke and transient ischemic attacks (TIAs). Recent studies proved the efficacy of this operation, compared with medical treatment for symptomatic high-grade stenosis (70–99%), symptomatic moderate stenosis (50–69%), and asymptomatic high-grade stenosis (≤ 60%).

The operation involves opening the common carotid artery and the proximal internal carotid artery in the neck (Fig 1.4-1), removing atherosclerotic plaque from the inside of the artery, and repairing the wall of the arteries (media and adventitia). Opening the carotid artery (**arteriotomy**) requires temporary occlusion of the proximal common carotid artery, distal internal carotid artery, external carotid artery and, usually, its first branch, the superior thyroid artery. The entire procedure can be achieved under continued occlusion of these vessels if the collateral blood flow to the territory supplied by the occluded internal carotid is deemed adequate (on the basis of intraop EEG monitoring, internal carotid artery back-bleeding, stump pressures, CBF studies, or angiography). Alternatively, an internal shunt between the proximal common carotid artery and distal internal carotid artery can be placed after the arteriotomy for use during the endarterectomy. Often a synthetic graft (e.g., Dacron) or, occasionally, a vein graft, is used to reconstruct (“patch”) the arteriotomy site and increase the luminal diameter.

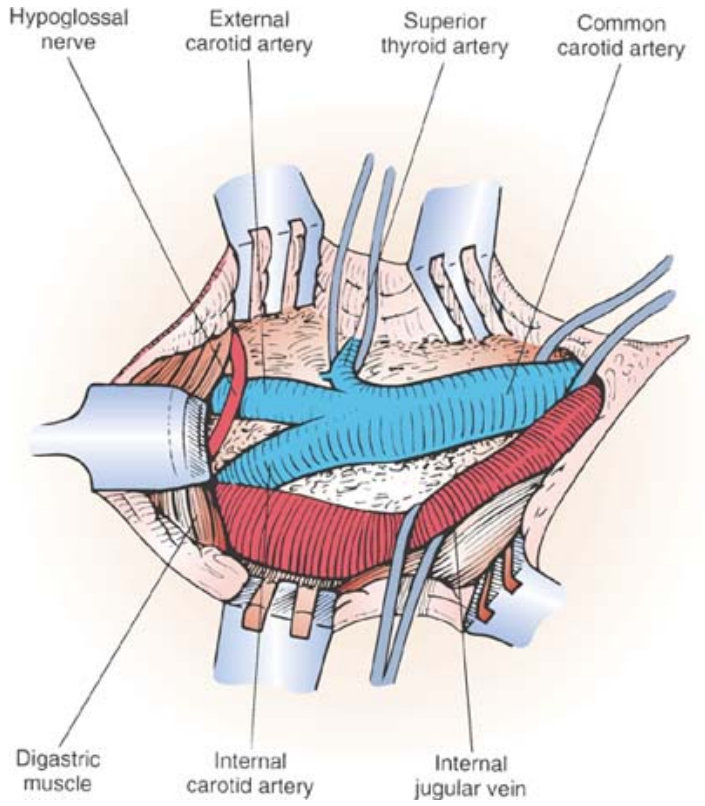

Figure 1.4-1. 1. Exposure and control of carotid artery. (Reproduced with permission from Calne R, Pollard SG: *Operative Surgery*. Gower Medical Pub: 1992.)

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Variant Procedure or Approaches: The vascular surgeon's approach to CEA is described in chapter 6.3. Carotid stenting is described in the Intracranial Neurology section (13.1). EC-IC Bypass for treatment of carotid occlusion is described in Section 1.1.

Usual preop diagnosis: Stroke; TIAs; carotid artery stenosis; carotid artery dissection

Summary of Procedures

Position	Supine
Incision	Anterolateral neck; occasionally, if "patching" arteriotomy, may have to harvest portion of greater saphenous vein from leg.
Special instrumentation	Magnification loupes; vascular instruments ± shunt (Bard, Javid, Pruitt-Inahara)
Unique considerations	Techniques for monitoring cerebral blood flow: EEG-spectral analysis or raw EEG, somatosensory evoked potentials (SEPs), back-bleeding or internal carotid artery stump pressure (> 50 mmHg), CBF measurements using Xenon, transcranial Doppler. Full anticoagulation with heparin (typically 7500–10,000 U iv) during arterial occlusion ± reversal with protamine (typically 35–50 mg iv) 10 min after repair and reopening of carotid arteries. Maintaining mild HTN during internal carotid artery occlusion (MAP 90–110). Use of intraop neuroprotective agents during internal carotid artery occlusion (e.g., iv STP 2–5 mg/kg to produce EEG burst-suppression).
Antibiotics	Cefazolin (1 g iv q 6 h)
Surgical time	3 h

Closing considerations	Avoid ↑BP or ↓BP (typical MAP 80–100); meticulous hemostasis.
EBL	50–150 mL
Postop care	Control of BP (MAP 80–100 mmHg); start aspirin on postop d 1; ICU or other monitored bed × 6–24 h.
Mortality	0.3–1.1% (combined CABG/CEA: up to 17.7%) 0.5–4% with postop MI
Morbidity	Cranial nerve injury: Up to 39% (Typically 8%) Stroke: 1–3% (2/3 postop) MI: 1–2%Postop bleeding: 1.7–2.7%Wound infection: Rare
Pain score	3

Patient Population Characteristics

Age range	50–90 yr
Male:Female	1:1
Incidence	150,000 CEAs/yr in the United States
Etiology	Atherosclerosis; occasionally, traumatic (dissection)
Associated conditions	HTN; CAD; PVD; smoking; obesity; alcohol abuse; hyperlipidemia

Anesthetic Considerations

(Procedure covered: carotid endarterectomy)

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Preoperative

The incidence of occlusive or ulcerative lesions of the extracranial or intracranial vasculature increases with advancing years. Generally, these lesions are asymptomatic until the cross-sectional area of the vessel is decreased by at least 50%. This is because in most patients the cerebral vasculature has excellent collateral circulation, most importantly the Circle of Willis (normal in only 50% of patients), but also the carotid-basilar anastomosis via the trigeminal artery and the extra- to intracranial collateral flow via the ophthalmic artery or branches of the vertebral artery. Patients presenting for CEA generally fall into one of three categories: (1) Those with TIAs, presenting with symptoms that may be focal or generalized. (2) Patients with completed stroke. If the stroke is recent (< 2–4 wk), some surgeons will not operate on the patient for fear of converting an ischemic infarct into a hemorrhagic infarct; however, if the infarct is small and clinical deficit minor, early surgery may be indicated. Angiographic evaluation usually demonstrates a stenotic and/or ulcerative lesion at the carotid bifurcation. (3) Patients with asymptomatic bruit, which usually is found during a routine physical examination of the neck. These are of concern because they may signal the development of carotid stenosis and may benefit from surgical intervention.

Respiratory	Patients should be asked to stop smoking prior to anesthesia, even if only the night before. Although cessation of smoking for such a short time will not lessen the volume of secretions appreciably or make the airways less irritable to a foreign body, such as an ETT, it will provide sufficient time for the carbon monoxide levels in the blood to decrease, thereby enhancing O_2carrying capacity. Any Hx of pulmonary disease should be evaluated with spirometry, ABGs, and CXR. If there is evidence of pulmonary infection, appropriate antibiotic therapy should be instituted. If secretions are excessive, preop pulmonary physiotherapy, including bronchodilator therapy, may be indicated. **Tests:** ABGs; spirometry; CXR, if indicated from H&P.

Cardiovascular	Careful evaluation of cardiovascular status is essential and should include a detailed Hx of cardiovascular function and serial determinations of BP in both arms to establish the range of pressures that normally occur, and whether there are regional differences. If BP is different in the two arms, it should be measured intraop and postop in the arm with the higher values. Also, a preop ECG is mandatory. The reason for concern about cardiovascular function is twofold: (1) It is often necessary to administer vasoactive drugs to regulate BP during CEA, either to maintain it at a normal value, or sometimes to increase it as much as 20% above the highest resting pressure to maintain optimal collateral circulation during surgical carotid occlusion. (2) The incidence of perioperative MI in this surgical population is > 1%, and represents the most common major postop complication in this operation. Except in the case of emergencies, anesthesia and operation should not proceed in the face of severe, uncontrolled HTN, diabetes, or recent MI. Antihypertensive medications should be continued up to the time of anesthesia. **Tests:** ECG; others as indicated from H&P.
Neurological	The Sx of cerebrovascular insufficiency are due to either critical stenosis or occlusion of cerebral vessels, combined with inadequate collateral circulation. The degenerative plaques or mural thrombi readily break off from the vessel wall and cause focal ischemic lesions. Manual occlusion of the carotid arteries is not an appropriate test of tolerance to temporary circulatory occlusion, as it may endanger the patient by precipitating embolization from an ulcerative lesion or by inducing bradycardia and ↓BP from activation of the carotid sinus reflex. It is desirable, however, to position the patient's head in the operative position as a test of the effect of that position on CBF. It is well-documented that hyperextension and lateral rotation of the head may occlude vertebral-basilar flow and, if sustained, contribute to postop cerebral ischemia. Sx of dizziness or diplopia will emerge with this maneuver if CBF is compromised. **Tests:** Carotid and cerebral angiography will identify type of lesion (ulcerative or stenotic), its location, and extent of collateral circulation. Other commonly used techniques include MR angiography, CT angiography, and duplex ultrasonography. As part of the preop evaluation, the anesthesiologist should examine the angiograms of the patient or discuss the case with the surgeon in order to understand with type, location, and extent of the lesion.
Hematologic	Aspirin or other anti-Plt therapy usually is begun preop to ↓ the risk of periop thromboembolic complications. **Tests:** PT; PTT
Laboratory	Tests as indicated from H&P.
Premedication	Use of premedication in patients undergoing CEA is controversial. Should a new TIA or stroke occur in the immediate preop period, its Sx may be difficult to distinguish from those associated with excessive responses to premedication. In this population, detailed discussion about the anesthetic and surgical plan, with appropriate reassurance, is usually sufficient. If medication is desired, midazolam 1–3 mg is preferable to opiates.

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Intraoperative

Anesthetic technique (regional): Historically, regional anesthesia—in the form of superficial and deep cervical plexus blocks, supplemented as needed by a local field block—was used for most CEAs. It provided the opportunity to evaluate cerebral function during a trial occlusion of 2–3 min. If the patient showed no adverse effects, the operation was completed under regional anesthesia. If the patient developed neurological changes, a shunt was inserted or GA was induced and ET intubation performed, following which the operation was completed. Regional anesthesia, however, has several disadvantages: absence of cerebral protection, patients may tolerate carotid occlusion for 10 min or more before suddenly losing consciousness or developing a Sz; and conversion to GETA may be technically difficult. Nevertheless, the technique still has some advocates among anesthesiologists and surgeons. It is claimed that: (1) it decreases the need for a surgical shunt and, thereby, avoids the complications of shunt insertion; (2) it decreases the length of stay in the ICU; and, (3) as an anesthetic technique, it is well-accepted by some patients.

Anesthetic technique (GETA): GETA offers both direct and indirect advantages for patients undergoing CEA: cerebral protection by decreasing $CMRO_2$ and redistributing flow toward the potentially ischemic area; greater patient comfort; and the ability to regulate PO_2, PCO_2, and MAP. Despite these arguments favoring GA, recent studies comparing regional and GA suggest that there is no clear outcome advantage of one technique over the other.

Induction	Both STP (3–5 mg/kg) and propofol (1–2 mg/kg), when carefully titrated, are suitable for induction agents, as arterial BP will generally remain at acceptable levels (± 20% of baseline) in normovolemic patients. These agents will ↓ $CMRO_2$ constrict normally reactive cerebral vessels, and → a redistribution of CBF toward potentially ischemic areas. Etomidate (0.1–0.4 mg/kg) may be useful for induction of anesthesia in hemodynamically unstable patients. A muscle relaxant, such as vecuronium (0.15 mg/kg), cisatracurium (0.1–0.2 mg/kg), or rocuronium (0.5–0.6 mg/kg) is administered for tracheal intubation. An analgesic, such as fentanyl (2–5 mcg/kg), or remifentanil (1–3 mcg/kg), may be given to minimize the cardiovascular responses to ET intubation. These opiates have minimal effects on CBF or $CMRO_2$
Maintenance	Isoflurane (up to 0.6%) or sevoflurane (up to 1%) inspired, in combination with N_2O 50% and remifentanil (0.05–0.2 mcg/kg/min), is satisfactory and will not interfere with EEG or EP monitoring. Just before cross-clamping of the carotid artery, an additional dose of STP sufficient to produce burst suppression on the EEG (usually 1–3 mg/kg) may be administered for its cerebral protective effects. Frawley et al. have suggested that STP adequately protects the brain during CEA, and that a surgical shunt is obsolete and not needed.
Emergence	Upon removal of the carotid cross-clamps, total carotid occlusion time should be noted on the anesthetic record. After 10 min and once bleeding from the arteriotomy site has been controlled, protamine (typically 0.5 mg/100 U heparin) is administered iv slowly over at least 10 min. If ↓BP occurs, rate of protamine administration should be slowed. If vasopressors were used during surgery, patient should be weaned from them during emergence, because HTN is likely as patient awakens from anesthesia. The need to control BP with a combination of esmolol and SNP/NTG is likely in the emergence phase. Antiemetic prophylaxis (e.g., ondansetron 4 mg iv 30 min before end of case) is usually appropriate. Prophylactic β-blockers may be quite useful in this patient population.

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Blood and fluid requirements	IV: 18 ga × 2 NS/LR @ 5–10 mL/kg/h	Blood replacement is seldom an issue. Studies indicate that elevated blood glucose may ↓ tolerance of brain to ischemia; thus, it is prudent to avoid glucose-containing solutions and treat levels > 180 mg/dL. One iv is dedicated to drug infusions.

	Hetastarch 6% < 20 mL/kg	If volume expansion is needed, hetastarch stays in the intravascular compartment longer (2–3 d) than crystalloid solutions (1–3 h).
Monitoring	Standard monitors (see p. B-1). Arterial line (often pre-induction) UO CVP (if otherwise indicated)	Marked fluctuations in BP may occur, necessitating vasoactive drug therapy. The arterial pressure transducer should be placed at the level of the head to accurately assess CPP. A CVP catheter is seldom necessary. Vasoactive drugs usually can be given safely through a second iv placed in the proximal arm. A second pressure transducer set-up may be needed for stump pressure measurements. Monitors incorporating EASI ECG analysis technology can provide 12-lead ECGs using only five actual leads.
	rCBF	Regional CBF (rCBF) ≤ 25 mL/min/100 g brain is satisfactory, and < 20 mL/min/100 g indicates potential for cerebral ischemia; however, capability for making rCBF measurements is not generally available in OR.
	EEG	A variety of spectral compression techniques permit computerized EEG analysis in OR. The major disadvantages are: the EEG may not identify small focal areas of ischemia; the depth of anesthesia and arterial CO_2must be stable or the EEG will not be interpretable; and there is a high incidence of false positives and negatives.
	Stump pressure	Stump pressure (pressure distal to the carotid clamp—also called “back pressure”— is used to evaluate the adequacy of cerebral perfusion. Cerebral ischemia rarely occurs at stump pressures > 60 mmHg. The major criticism of stump pressure is the large number of false positives. This occurs in 1/3 of patients and results in a shunt being placed when none is needed. The simplicity of the measurement and its relative validity when pressure is > 60 mmHg still make it a useful clinical method for ensuring adequate perfusion.
Cerebral perfusion monitoring	Cerebral oximetry	Cerebral oximetry (e.g., Somanetics Invos) is being used with increasing frequency to evaluate cerebral perfusion during CEA. Cerebral oximeter sensors are placed on the forehead, where it is presumed they measure O_2sat in the superficial frontal cortex. With carotid occlusion, ipsilateral oximetric values may decrease suggesting inadequate cerebral perfusion. The method is nonquantitative and a portion of the signal may originate from structures superficial to the cortex perfused by branches of the external carotid artery. False positive and false negative results can

		occur.
	SSEP	SSEPs are being used with increasing frequency as a means of determining the adequacy of cerebral perfusion during temporary occlusion of the carotid artery. This technique requires continuous monitoring by personnel with special training. Areas of focal ischemia may not be detected. False positive and false negative results can occur.
	Transcranial Doppler (TCD)	TCD scanning alone or combined with EEG monitoring is a useful method for detecting microemboli (air or particulate matter) during CEA. It also has been suggested that TCD can be used as a guide for regulating BP postop to minimize the occurrence of post-CEA cerebral hyperperfusion states. This technique requires continuous monitoring by personnel with special training, and may be technically difficult to implement intraop.
Control of BP	Keep MAP ≤ awake levels. Autoregulation Vasopressors: Phenylephrine infusion	It is highly desirable to maintain MAP at or slightly above the patient's highest recorded resting pressure while awake. Volatile anesthetics impair autoregulation; therefore, the higher the pressure, the more likely it is that cerebral perfusion will be adequate during surgical occlusion. A pure a-adrenergic agonist (e.g., phenylephrine) is ideal to support BP, because it has minimal dysrhythmogenic potential. A V-4 or V-5 lead usually will indicate if ↓ BP is causing myocardial ischemia.
	Coronary vasodilation NTG infusion	A simultaneous infusion of NTG (0.2–0.4 mcg/kg/min) may improve coronary flow during periods of induced ↑ BP. If hypertensive episodes occur during surgery, infusions of esmolol ± NTG/SNP work well to control BP.
	Labetalol (pre-emergence)	Long duration effect makes intraop titration difficult
Positioning	and pad pressure points. eyes.	

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Postoperative

	Circulatory instability: ↓ BP	Circulatory instability is common. ↓ BP may be due to hypovolemia, depression of circulation by anesthetic or other drugs, dysrhythmias, or exposure of the baroreceptor mechanism to a new higher pressure, causing an exaggerated reflex response. Rx by volume expansion and pressors.

	HTN MI Stroke	HTN may be due to loss of the normal carotid baroreceptor mechanism. It may → excessive bleeding, increased myocardial O_2 consumption, dysrhythmias, MI, intracerebral hemorrhage and ↑ICP from cerebral edema.
	Loss of carotid body function	Ipsilateral chemoreceptor function is lost in most patients after CEA, → ↓ventilatory and circulatory responses to hypoxia and a modest increase in resting arterial $PaCO_2$ These patients should be given supplemental O_2 postop. Special attention must be directed toward preventing atelectasis or other pulmonary or circulatory abnormalities that might cause hypoxemia.
Complications		
	Respiratory insufficiency	Acute respiratory insufficiency may occur 2° hematoma formation with tracheal deviation, vocal cord paralysis from surgical traction on laryngeal nerves, or tension pneumothorax from dissection of air through the wound into the mediastinum and pleural space. Unexpected respiratory distress should immediately bring these three possibilities to mind, with appropriate Dx and therapy. A hematoma that causes respiratory distress always should be evacuated before reintubation is attempted.
	Tension pneumothorax	If there is evidence of circulatory insufficiency, a tension pneumothorax should be relieved immediately by needle evacuation.
	Intimal flap → stroke	Should a patient emerge from anesthesia with a new neurological deficit, immediate surgical exploration of the operative site or immediate cerebral angiography should be performed to determine if an intimal flap has formed at the site of operation. This is a surgically correctable lesion and, if corrected immediately, may lessen the severity of the subsequent neurological deficit.
Pain management	Meperidine (10 mg iv prn) Codeine (30–60 mg im q 4 h)	
Tests	Cerebral angiography Carotid ultrasound	

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Suggested Readings

1. Allen BT, Anderson CB, Rubin BG, Thompson RW, Flye MW, Young-Beyer P: The influence of anesthetic technique on perioperative complications after carotid endarterectomy. *J Vasc Surg* 1994; 19:834–42.

2. Archer DP, Tang TKK: The choice of anaesthetic for carotid endarterectomy: does it matter? *Can J Anaesth* 1995; 42:566–70.

3. Fiori L, Parenti G, Marconi F: Combined transcranial Doppler and electrophysiologic monitoring for carotid endarterectomy. *J*

Neurosurg Anesthesiol 1997; 9:11–6.

4. Frawley JE, Hicks RG, Gray LJ, Niesche JW: Carotid endarterectomy without a shunt for symptomatic lesions associated with contralateral severe stenosis or occlusion. *J Vasc Surg* 1996; 23:421–7.

5. Guay J. Regional or general anesthesia for carotid endarterectomy? Evidence from published prospective and retrospective studies. *J Cardiothorac Vasc Anesth* 2007; 21(1):127–32.

6. Kearse LA Jr, Lopez-Bresnahan M, McPeck K, Zaslavsky A: Preoperative cerebrovascular symptoms and electroencephalographic abnormalities do not predict cerebral ischemia during carotid endarterectomy. *Stroke* 1995; 26:1210–4.

7. Luebke T, Aleksic M, Brunkwall J. Meta-analysis of randomized trials comparing carotid entarterectomy and endovascular treatment. *Eur J Vasc Endovasc Surg* 2007; 34(4):470–9.

8. Mutch WAC, White IWC, Donin N, Thomson IR, Rosenbloom M, Cheang M, West M: Haemodynamic instability and myocardial ischaemia during carotid endarterectomy: a comparison of propofol and isoflurane. *Can J Anaesth* 1995; 42:577–87.

9. Sajid MS, Vijaynager B, Singh P et al.: Leterature review of cranial nerve injuries during carotid endarterectomy. *Acta Surg Belg,* 2007; 107(1): 25–8.

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10. Sejersten M, Wagner GS, Pahlm O et al.: Detection of acute ischemia from the EASI-derived 12-lead electrocardiogram acquired in clinical practice. *J Electrocardiol* 2007; 40:120–6.

11. Wade, JG, Larson CP Jr, Hickey RF, Ehrenfeld WK, Severinghaus JW: Effect of carotid endarterectomy on carotid chemoreceptor and baroreceptor function in man. *N Engl J Med* 1970; 282(15):823–9.

12. Warner DS, Hindman BJ, Todd MM, Sawin PD, Kirchner J, Roland CL, Jamerson BD: Intracranial pressure and hemodynamic effects of remifentanil versus alfentanil in patients undergoing supratentorial craniotomy. *Anesth Analg* 1996; 83:348–53.