Chapter 10
Carotid Endarterectomy

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10.1 Introduction

Stroke is the second leading cause of death worldwide. One of the main causes of stroke is carotid artery stenosis. Stenosis with atherosclerosis in the carotid artery can cause stroke by hemodynamic ischemia or artery to artery embolism. Carotid artery stenosis has been often treated with surgical interventions. Carotid intervention was first successfully performed in 1951 by excision of the diseased carotid artery segment and an end-to-end anastomosis of internal carotid artery and common carotid artery [1, 2]. Since then, carotid endarterectomy (CEA) has been evolved with introduction of temporary shunt system in 1956 [3], eversion endarterectomy in 1970 [4], and electroencephalogram monitoring in 1980 [5]. In 1980s and 1990s several randomized control trials (RCTs) have proven efficacy of CEA compared to medical treatment in symptomatic and asymptomatic patients [6–11]. Since carotid artery stenting (CAS) was appeared in 1990s, several RCTs has been conducted [12–18]. As the devices have been developed, treatment outcomes of CAS have been improved, and CAS has been shown to be equally beneficial to CEA with some conditions [17, 18]. By its curability and long-stand stroke preventive effect, however, CEA is still first choice of treatment for symptomatic severe carotid stenosis [19]. Here, we review the recent RCTs for CEA, explain the perioperative management, and show surgical techniques with illustrations.

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Y. Kato, A. Ansari (eds.), *Cerebrovascular Surgery*, Advances and Technical Standards in Neurosurgery 44, https://doi.org/10.1007/978-3-030-87649-4_10

10.2 Evidence of CEA

10.2.1 CEA for Symptomatic Carotid Stenosis

For symptomatic carotid stenosis, two large RCTs, European Carotid Surgery Trial (ECST), and North American Symptomatic Carotid Endarterectomy Trial (NASCET) compared CEA with medical treatment.

In ECST, 3024 carotid stenosis patients with transient or mild symptomatic ischemic vascular event on the distribution of one or both carotid arteries were allocated to medical treatment only or CEA. As a result, ipsilateral stroke or perioperative death with more than 80% stenosis was 20.6% in medical group and 6.8% in CEA group (<0.0001) [8].

NASCET was started in 1987 in North America. Patients who experienced transient ischemic attack (TIA) or nondisabling stroke within 120 days were assigned to optimal medical care alone or optimal medical care plus CEA. The results showed that cumulative risk of any ipsilateral stroke at 2 years with 70–99% stenosis were 26% in medical group and 9% in surgical group ($P < 0.001$) on the condition that the treatments were conducted in the centers with the rate of less than 6% for stroke and death occurring within 30 days of operation [6]. Also, the ipsilateral stroke risk at 5 years with 50–69% stenosis were 22.2% in medical group and 15.7% in surgical group, whose difference became statistically significant ($P = 0.045$) if the surgeons have lower rates of complications than 2% [7]. Efficacy of CEA in symptomatic patients with less than 50% stenosis has not been proved.

Mata-analysis of ECST and NASCET focused on clinical subgroups and timing of surgery was reported in 2004 [20]. CEA was especially beneficial in men, patients aged 75 years or older, and patients who underwent surgery within 2 weeks of their last symptoms, and fell rapidly with increasing delay (see Timing of Surgery).

10.2.2 CEA for Asymptomatic Carotid Stenosis

For asymptomatic carotid stenosis, Asymptomatic Carotid Atherosclerosis Study (ACAS) began in 1987. Medical treatment and CEA were compared in 1662 patients with asymptomatic carotid stenosis of 60% or greater. The aggregate risk over 5 years for ipsilateral stroke and any perioperative stroke or death was 11.0% in medical group and 5.1% in CEA group ($P = 0.004$), which proved the efficacy of CEA if it was performed with less than 3% perioperative morbidity and mortality [9].

Another trial for asymptomatic stenosis, Asymptomatic Carotid Surgery Trial (ACST), was started in 1993. It compared deferral of any carotid procedure and immediate CEA in asymptomatic patients with at least 60%. The risk of perioperative events and strokes was 10.9% in deferral CEA group and 6.9% in immediate CEA group at 5 years ($P = 0.0001$) and 17.9% and 13.4% at 10 years ($P = 0.009$)

[10, 11]. Contrary to the trials in symptomatic patients, these ACST showed no significant association between the risk of stroke and the percentage of stenosis, and CEA was effective for both males and females.

With a recent progress of intensive medical therapy, however, the superiority of CEA to medical therapy becomes equivocal in asymptomatic patients [21]. In practice, CEA is recommended only when the stenosis is more severe (70–99%) or the patients have a particular high risk of stroke (progression of stenosis, the detection of asymptomatic carotid embolism, carotid plaque vulnerability, reduced cerebrovascular reserve, and the presence of silent embolic infarcts) [22]. Other than these risk factors, we should consider the comorbidities and life expectancy of the patients and also the surgeons' experience so as to get maximum benefit from CEA. In our opinion, the indication of CEA for asymptomatic patients should depend not only upon guidelines, but also upon the tailor-made medicine.

Updated guidelines for the treatment of carotid stenosis from American Heart Association (AHA), Society of Vascular Surgery (SVS), and European society of vascular surgeons (ESVS) are listed in Table 10.1 [23–26].

10.2.3 CEA vs. CAS

Since CAS was first performed in 1994, several RCTs comparing CEA and CAS have been reported. The first RCT comparing CAS to CEA was Stent and Angioplasty with Patients at High Risk for Endarterectomy (SAPPHIRE) trial [13]. The trial focused on the patients at high risk for CEA who have at least one of the following risk factors: positive stress test; age older than 80 years; contralateral carotid occlusion; pulmonary dysfunction; high cervical lesion; repeat carotid operation; congestive heart failure and/or known severe left ventricular dysfunction; open heart surgery needed within 6 weeks; recent myocardial infarction; unstable angina; contralateral laryngeal nerve palsy; radiation therapy to the neck. In this trial, CAS is proved not to be inferior to CEA at 1 year and also at 3 years' follow-up [13]. For patients without high risk for CEA, Endarterectomy Versus Angioplasty in Patients with Symptomatic Severe Carotid Stenosis Trial (EVA-3S) [14], Stent-Protected Angioplasty versus Carotid Endarterectomy (SPACE) [15], and International Carotid Stenting Study (ICSS) [16] compared CAS and CEA for symptomatic stenosis. All of these three trials failed to show the non-inferiority of CAS to CEA. We need to note that these three trials did not require the use of protection devices in CAS, and the surgeons were not selected strictly. Contrary to these three trials, recent RCTs reported the equal benefit of CEA and CAS. One of these RCTs is Carotid Revascularization Endarterectomy versus Stenting Trial (CREST). The trial compared CAS with embolic protection devices and CEA in symptomatic patients (>50% stenosis), and asymptomatic patients (>70% stenosis). There was no significant difference in the primary end point: 4-year rates of stroke, myocardial infarction, or death of any cause during the periprocedural period or any ipsilateral stroke within 4 years after randomization (7.2% in CAS and 6.8% in

Table 10.1 Guidelines of CEA

	AHA guidelines (2014 for symptomatic, 2011 for asymptomatic)	SVS guidelines (2011)	ESVS/ESC guidelines (2017)
Symptomatic	For patients with a TIA or ischemic stroke within the past 6 months and ipsilateral severe (70–99%) carotid artery stenosis as documented by noninvasive imaging, CEA is recommended if the perioperative morbidity and mortality risk is estimated to be <6%. (class I, level of evidence A)	In most patients with carotid stenosis who are candidates for intervention, CEA is preferred to CAS for reduction of all-cause stroke and periprocedural death (grade I, level of evidence B).	CEA is recommended in symptomatic patients with 70–99% carotid stenosis, provided the procedural death/stroke rate is <6% (class I, level of evidence A).
	For patients with a TIA or ischemic stroke within the past 6 months and ipsilateral moderate (50–69%) carotid artery stenosis as documented by catheter-based imaging or noninvasive imaging with corroboration (e.g., magnetic resonance angiogram or computed tomography angiogram), CEA is recommended depending on patient-specific factors like age, sex, comorbidities, if the perioperative morbidity and mortality risk is estimated to be <6% (class I, level of evidence B).	Data from CREST suggest that patients aged <70 years may be better treated by CAS, but these data need further confirmation.	CEA should be considered in symptomatic patients with 50–69% carotid stenosis, provided the procedural death/stroke rate is <6% (class IIa, level of evidence A).

Table 10.1 (continued)

	AHA guidelines (2014 for symptomatic, 2011 for asymptomatic)	SVS guidelines (2011)	ESVS/ESC guidelines (2017)
Asymptomatic	Selection of asymptomatic patients for carotid revascularization should be guided by an assessment of comorbid conditions, life expectancy, and other individual factors and should include a thorough discussion of the risks and benefits of the procedure with an understanding of patient preferences (class I, level of evidence C).	Neurologically asymptomatic patients with equal or more than 60% diameter stenosis should be considered or CEA for reduction of long-term risk of stroke, provided the patient has a 3- to 5-year life expectancy and perioperative stroke/death rates can be equal or less than 3% (grade I, level of evidence A).	In "average surgical risk" patients with an asymptomatic 60–99% stenosis, CEA should be considered in the presence of clinical and/or more imaging characteristics that may be associated with an increased risk of late ipsilateral stroke, provided the perioperative stroke/death rates are <3% and the patient's life expectancy is >5 years (class IIa, level of evidence B).
	It is reasonable to perform CEA in asymptomatic patients with more than 70% stenosis if the risk of perioperative stroke, MI, and death is low. (class IIa, level of evidence A)		

CEA, $P = 0.51$), and the rates did not differ depending on symptomatic status ($P = 0.84$) or sex ($P = 0.34$). However, an interaction between age and treatment efficacy was detected ($P = 0.02$); CAS tended to show greater efficacy at younger than 70 years, and CEA at older. Moreover, periprocedural complication rate differed between CAS and CEA: the stroke rate was higher in CAS (4.1% and 2.3%, $P = 0.01$), and the myocardial infarction rate was higher in CEA (1.1% and 2.3%, $P = 0.03$). The rate of ipsilateral stroke was low in both groups (2.0% and 2.4%, $P = 0.85$) [17].

Another RCT showing efficacy of CAS is Asymptomatic Carotid Trial (ACT) one reported in 2016. The study targeted asymptomatic patients with at least 70% stenosis aged 79 years or younger without high risk for CEA, and compared CAS with embolic protection and CEA. In this study, CAS was noninferior to CEA for the prevention of ipsilateral stroke and death until 5-year follow-up period [18]. Today some new RCTs (CREST-2, ECST-2, and ACST-2) are now ongoing. In these trials, not only CEA vs CAS but also best medical treatment (BMT) vs interventions (CEA/CAS) are being compared. Indication of intervention should be reconsidered based on the upcoming trials' results.

10.3 Theoretical Background of CEA

Most of the carotid plaques are known to be limited to carotid bifurcation. The reason of this localization is not clear, but shear stress seems to play an important role in the formation of atheromatous plaque. In the carotid bifurcation, blood stream changes its direction, which can induce shear stress to the vessel wall [27–29]. In addition, Hori et al. reported that the characteristics of artery change from elastic to muscular artery at the bifurcation, and this histological change can also affect atheromatous formation [30]. They mentioned this change ended up to 20 mm distal from the bifurcation and plaque formation was also terminated up to 25 mm distal from bifurcation in most of the cadaver cases even with severe atherosclerosis.

Theoretically, in other words, we can remove almost all plaques with CEA when we expose distal ICA more than 25 mm from the bifurcation, although there are some exceptions.

10.4 Timing of Surgery

It is not well-known which timing is best for CEA after stroke or TIA. Concerning about TIA, risk for stroke onset after TIA increases by time, especially within 14 days. It has been reported that stroke occurs in 5–8% of patients with 50–99% carotid stenosis within 48 h after the index TIA, 4–17% within 72 h, 8–22% within 7 days, and 11–25% within 14 days [31–38], which indicate early (<14 days) intervention is beneficial to prevent stroke in TIA (or minor stroke) patients [20]. But the effectiveness of urgent (<24, or 48 h) CEA is still controversial. The 2017 Clinical Guidelines of European Society for Vascular Surgery states that patients with 50–99% stenosis who present with crescendo TIA should be considered for an urgent CEA, preferably within 24 h [39], but a systemic analysis demonstrated that CEA within 48 has a beneficial effect for crescendo TIA patients, but its effectiveness was not different between CEA within 24 h and after 24 h [40]. In addition, for the patients who have large infarct volume (≥1/3 of MCA territory) and severe disability (modified Rankin score ≥3), CEA should be deferred to minimize the risks of postoperative parenchymal hemorrhage [41]. Recent report shows urgent (<48 h) CEA leads to worse functional outcome if it is applied to the patients with moderate to severe strokes (NIHSS >10) [42].

In summary, the patients who have symptomatic 50–99% carotid stenosis should undergo CEA.

1. Within 14 days if the symptom is TIA or minor stroke and within 48 h is better if possible.
2. After 30 days if the symptom is severe (mRS ≥ 3 or NIHSS>10) or infarct volume is large (≥1/3 of MCA territory).

10.5 Imaging

10.5.1 Carotid Ultrasonography (CUS)

CUS is less invasive and suitable for screening. It can also evaluate plaque fragility by its echo-lucency. Hypoechoic (echo-lucent) plaque seems lipid-rich fragile plaque, whereas hyperechoic plaque seems elastic, and/or calcified stable plaque. Doppler-CUS gives us the peak systolic velocity (PSV) of the blood stream, which helps us to estimate the severity of stenosis. PSV > 125 cm/s means >50% stenosis, and PSV > 200–230 cm/s means >70% stenosis [43, 44].

10.5.2 Magnetic Resonance Imaging (MRI) and Angiography (MRA) (Fig. 10.1)

MRI/MRA is also less invasive imaging modality and gives us much more information about plaque characteristics with higher reproducibility than CUS. We can grasp the plaque extension, which is helpful to decide the range of distal ICA

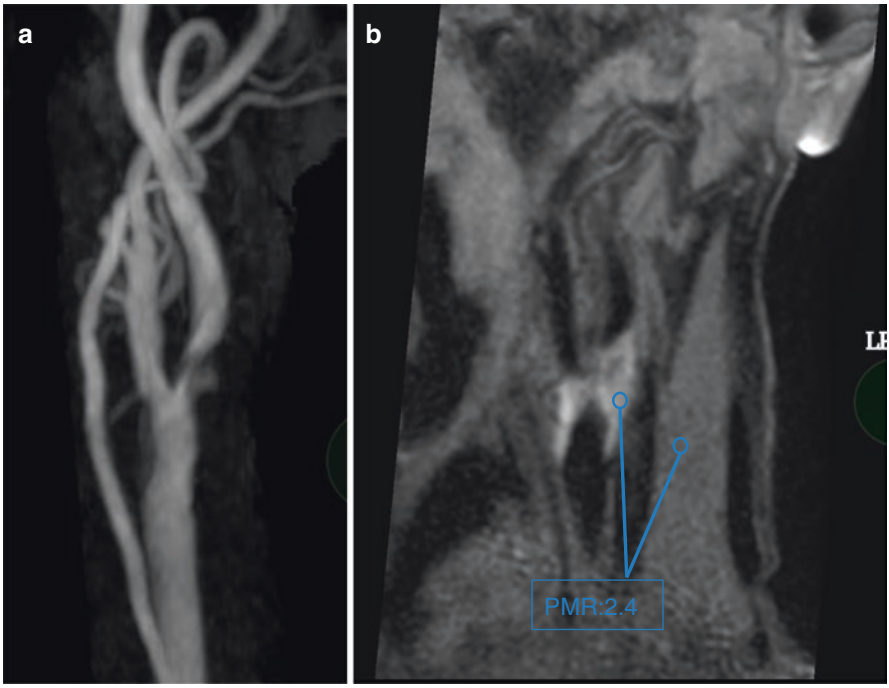

Fig. 10.1 Magnetic resonance angiography (MRA) of carotid artery. (**a**) time-of-flight (TOF) image. (**b**) black-blood (BB) image. In this patient, plaque/muscle ratio (PMR) was 2.4, which indicated fragile plaque

exposure during CEA [45]. With black-blood MRI (BB-MRI), moreover, plaque vulnerability can be evaluated. The plaque-to-muscle (sternocleidomastoid muscle) signal intensity ratio (plaque/muscle ratio [PMR]) is widely used and PMR > 3 is thought to be very fragile, PMR 1–3 be fragile, whereas PMR <1 is stable [46, 47].

10.5.3 Computed Tomography Angiography (CTA) (Fig. 10.2)

CTA needs contrast medium and X-ray exposure, which means more invasive than CUS and MRI, but much less invasive than digital subtraction angiography (DSA) because of no necessity of catheter procedure. Using three-dimensional (3D) CTA with bone images, surgical simulation becomes possible. We routinely measure mandibular angle to bifurcation length (M-B length) and according to this length, skin incision is designed in CEA.

10.5.4 Other Imaging Modalities

Conventional DSA is not always necessary for CEA patients, considering its risk. Brain MRI is to be done just prior to surgery to check the presence of fresh infarction in the ipsilateral brain and MRA is also to be done to check tandem lesion distal to the CEA site, and also to estimate the collateral blood flow from contralateral ICA or posterior circulation through circle of Willis during cross clamping. In our institute, cerebral blood flow (CBF) evaluation with single photon emission CT (SPECT) becomes mandatory to estimate the risk of postoperative cerebral hyperperfusion syndrome (CHS) if the patient seems to have hemodynamic compromise [48, 49]. The patients who have severe hypoperfusion preoperatively tend to suffer from CHS.

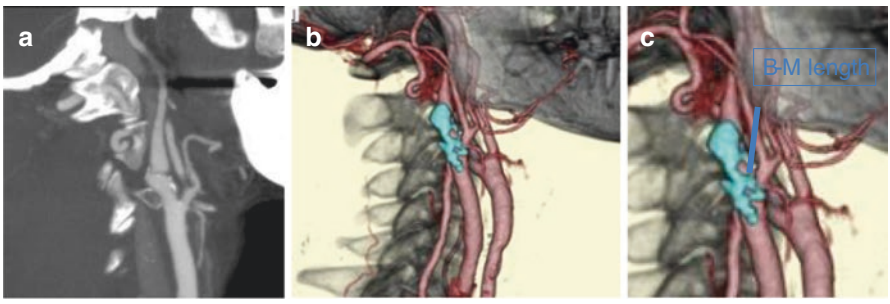

Fig. 10.2 Computed Tomography Angiography (CTA) of carotid artery. (**a**) Maximum intensity projection (MIP) image. (**b**) 3D-CTA shows anatomical landmarks around the lesion. (**c**) Mandibular-Bifurcation length (M-B length) is useful to estimate the bifurcation point before CEA

10.6 Anatomy of CEA (Fig. 10.3)

It is essential to know the surgical anatomy quite well before we start real surgery. In CEA, anatomy itself is not complicated. We do surgery inside the "carotid triangle," which is surrounded by three muscles (sternocleidomastoid, omohyoid, and posterior belly of digastric muscle), and other than arteries and veins, we should know the running course of two important nerves: hypoglossal nerve and superior laryngeal nerve. Descending branch of hypoglossal nerve (ansa cervicalis) could be cut without any symptoms, but a damage to superior laryngeal nerve can cause hoarseness and/or dysphagia. Different from hypoglossal nerve, branches of this nerve are very fine and cannot usually be identified during surgery, so comprehending the anatomy of this nerve and avoiding the rough dissection around this nerve (especially near the external carotid artery) lead to functional preservation.

10.7 Preoperative Management

10.7.1 Risk Management of General Anesthesia

Many patients who need CEA have some comorbidities such as other atheromatous vessel disease, pulmonary disease, and renal failure. In particular, those who have coronary artery disease (CAD) likely to develop myocardial infarction (MI) after

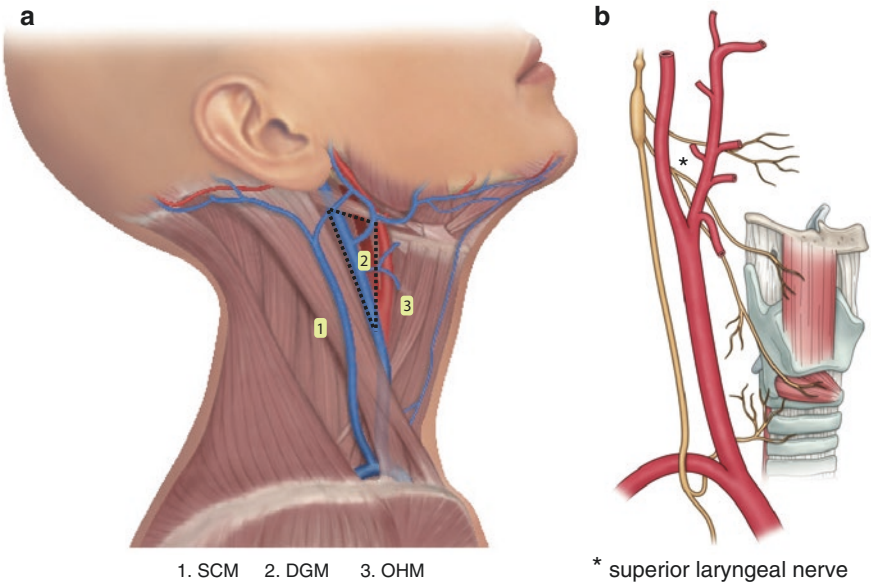

Fig. 10.3 Anatomy of CEA. (**a**) Carotid triangle is a triangle which was surrounded by three muscles. *SCM* sternocleidomastoid muscle, *DGM* digastric muscle, *OHM* omohyoid muscle. (**b**) Nerves around carotid arteries. Note the branches of superior laryngeal nerves run very close to external carotid and superior laryngeal nerve

CEA, so that careful checkup of CAD with ECG with physical load test, myocardial perfusion scintigraphy, or coronary 3D-CTA is necessary. If CAD coexists, intervention to it should take a priority if there is a time before CEA.

10.7.2 Antiplatelet Therapy

Cessation of antiplatelet therapy increases the risk of perioperative stroke and MI, so single or also dual antiplatelet therapy should continue just before CEA. On the other hand, anticoagulation therapy for atrial fibrillation can be stopped before surgery because the combination of antiplatelet and anticoagulation therapy may increase the risk of postoperative bleeding [50].

10.8 Neurophysiological Monitoring and Shunt Usage

Routine shunt or selective shunt usage during cross clamping is still controversial. Routine use of shunting system may increase the risk of intimal injury, dissection, and thrombosis formation by its insertion and removal [51], and shunt system disturbs the surgical view, which makes exposure of distal plaque end sometimes difficult. To select the patients who definitely require the shunt, neurophysiological monitoring becomes mandatory. Electroencephalography (EEG) [52, 53], Transcranial Doppler flowmetry (TCD) [54, 55], Near-infrared spectroscopy (NIRS) [56–58], Somatosensory evoked potential (SSEP) [59–61], and Motor evoked potential (MEP) [62, 63] have been applied as a single or multiple monitoring [64, 65] during CEA, and their efficacy has been reported for the selection of shunt-required patients and also for the prediction of the postoperative functional status. However, cut-off value of each monitoring has not been established. In our institute, multiple monitoring with EEG, SSEP, and MEP has been used. To our impression, EEG change occurs rapidly after cross clamp, but it cannot be quantified and sometimes recovers spontaneously. Therefore, we use EEG change as an alert of hypoperfusion, and if it is followed by the SSPE and/or MEP changes (cut-off value <50%), internal shunt is applied. Under this multiple monitoring, the incidence of shunt usage is approximately 10% without false negative.

10.9 Standard Surgical Procedure (Figs. 10.4–10.7)

We commonly use general anesthesia. The patients who have high carotid bifurcation needs nasal intubation. To lift up the mandibular angle, neck tends to be extended with vertex down position, but it has a risk to worsen the cervical spondylosis (CS). Those who have a history of CS or have myelopathic symptoms in the

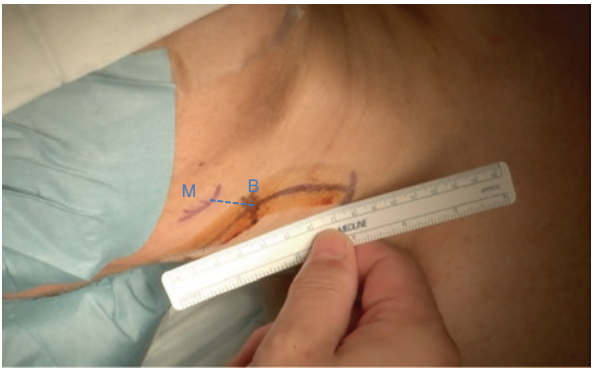

Fig. 10.4 Skin incision. According to the B-M length in 3D-CTA, carotid bifurcation is marked first. Skin incision is made 4 cm above and 4 cm below this point (total 8 cm)

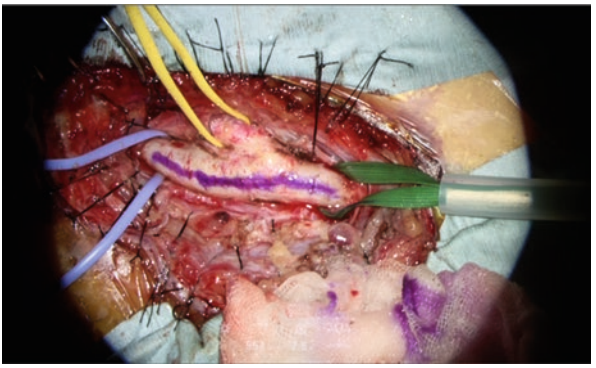

Fig. 10.5 Exposure of carotid arteries with hitch-up method. Note carotid sheath was hitched up to the surface, which makes retractors unnecessary. Carotid artery is exposed more than 2.5 cm distal and 2 cm proximal from the bifurcation in all cases

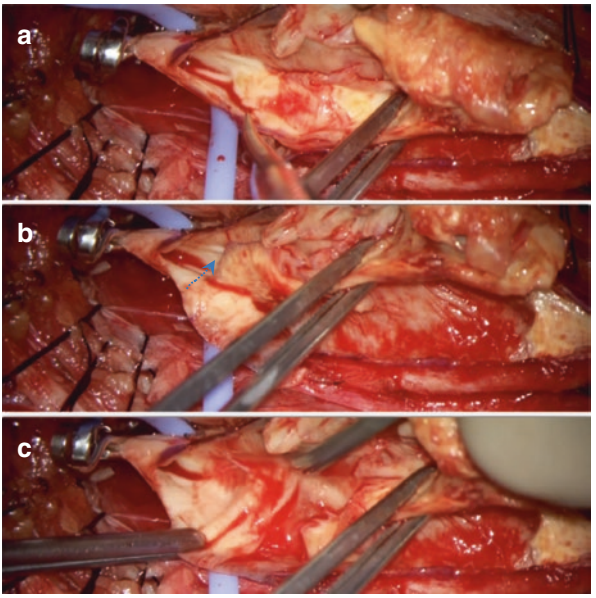

Fig. 10.6 Removal of the plaque at the distal end. (**a**): Sharp cut is sometimes necessary at the border of plaque and normal intima. (**b**) Plaque should be dissected toward vertical direction (*dotted arrow*). (**c**) After plaque removal. Note no intimal flap was made, which makes taking suture unnecessary

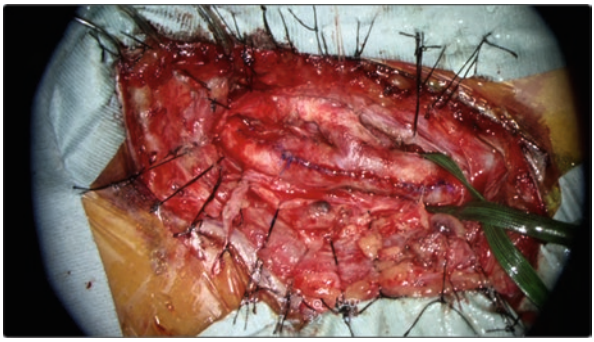

Fig. 10.7 Primary closure with 6-0 Nylon. Small suture bite-to-stich interval in the ICA is important to prevent postoperative restenosis

preoperative neck extension test, we should not keep the patients in the vertex down position. Instead, we can get similar surgical working space by lifting up the mandibular bone with blunt hooks even in the normal head positioning.

According to B-M length measured by the preoperative CT angiography, first we mark the carotid bifurcation on the skin. Skin incision is made 4 cm above and 4 cm below the marking of bifurcation (total 8 cm) along with the anterior margin of sternocleidomastoid muscle (SCM). After cutting the skin and platysma muscle, SCM is exposed. First, dissection is to be done between SCM and omohyoid muscle to expose common carotid artery (CCA). Anterior margin of SCM is hitched up laterally to expose the carotid sheath, and internal jugular vein is not always to be exposed. Carotid sheath is cut and hitched upward together with carotid arteries to get shallow surgical field. Common facial vein is cut during this exposure, but care should be taken no to cut the hypoglossal nerve which sometimes runs very close to this vein. How to identify the hypoglossal nerve is to follow the ansa cervicalis or to dissect just below the posterior belly of digastric muscle, which could easily be found just below the parotid gland. The patient who has high carotid bifurcation requires us some effort to expose the distal end of plaque. We have used tailor-made mouth piece to achieve mandibular subluxations [66] but recently it is thought to be enough to dissect SCM and parotid gland as much as possible and lift up the mandible with blunt hooks with nasal intubation. We commonly expose the carotid artery at least 2.5 cm distal to the bifurcation, 2 cm proximal to it according to the literature mentioned above [30], but it can be modified by refereeing the plaque imaging in MRA [45]. To visualize the distal end of the plaque clearly even in case of shunt usage, we think 5 mm more to be exposed from the distal edge of the plaque, and more exposure leads to less possibility of acute postoperative occlusion of distal ICA. After systemic heparinization with activated clotting time (ACT) >250 s, cross clamp is made. According to the intraoperative monitoring, we selectively use internal shunt. The plaque is removed from CCA to ICA. ECA plaque is easily pulled out without additional arteriotomy, but ICA plaque end should not be pulled out blindly. With adequate exposure, ICA plaque end must be visually

confirmed and removed totally with gentle dissection. Most of the plaques can be dissected from normal intima at their ends by splitting the margin with micro-scissors and move the plaques laterally. Tacking suture is done with 6-0 nylon only when the intimal flap formation is recognized at the distal end of dissection (rare). Vessel wall is closed with 5-0 Nylon running suture from both sides and overlapped 5 mm at the midpoint of suture line. Prior to total declamping, ECA and CCA clips are released temporally to prevent air embolism to ICA and secure the hemostasis. Additional stitches are requested when the arterial bleeding occurs from suture line, but small oozing can be stopped with gentle compression and heparinization reversal (after total declamping), or hemostatic agent (Floseal®). After checking the patency of ICA with Doppler sonography or flowmetry, wound is closed with layer-by-layer.

10.10 Controversial Issue

10.10.1 Eversion or Standard CEA?

Eversion CEA is first described by DeBakey et al. in 1959 [67]. This technique is known to be superior to standard CEA in terms of short surgical time and less frequent restenosis [68]. Instead, shunt insertion is more challenging and access to high lesion is difficult. Moreover, eversion CEA requires full dissection around carotid bulb and distal ICA that may lead to cranial nerve palsies. We prefer standard CEA because Asian people usually have high carotid bifurcation and shunt insertion is requested for approximately 10% of patients in our series with multiple neurophysiological monitoring.

10.10.2 Primary Closure or Patch Angioplasty?

Primary closure is a simple method and can reduce the clamp time but may increase the incidence of acute occlusion or restenosis. Patch angioplasty is thought to reduce these complications even though longer operation time and rare complication of vein graft rupture and patch infection were reported [69–71]. Patch material is made from an autologous vein, bovine pericardium, or synthetic material including polytetrafluoroethylene (PTFE), dacron, polyurethane, and polyester. The difference of patch material does not affect the outcome so much [72]. In our institute, patch angioplasty is not mandatory because we have rarely encountered restenosis after CEA (1%) with primary closure. We have used as small suture bite-to-stich interval as possible and tried not to involve the adventitia in the suture, which may lead to prevent acute occlusion or restenosis (Fig. 10.7). On the other hand, those who have originally small diameter in ICA (especially women) or CEAs after

restenosis are treated with patch angioplasty (Hemashield patch graft), so this technique should be ready to use whenever necessary.

10.10.3 Restenosis After CEA

It has been reported that restenosis occurs in 5–22% after CEA [73–75]. Restenosis is defined as more than 50% of stenosis after more than 30 days postoperatively [76]. Pathophysiology of early restenosis (within 2 years after CEA) is thought not to be atherosclerosis, but to be inflammation and neo-intimal hyperplasia, so it is not likely to cause artery to artery embolism even though the stenosis becomes severe. But late restenosis (>2 years after CEA) is deemed similar to primary atherosclerotic lesion that can become an embolic source [77]. There is no clear guideline to treat post-CEA restenosis, but controlling the risk factors is most essential. Vascular risk factors (hyperlipidemia, hypertension, smoking, and metabolic syndrome), and female gender have been described as risk factors [75], so best medical treatment (BMT) should continue and careful follow-up is necessary to the patients who have those factors. If the restenosis becomes severe (>70%) and symptomatic, reintervention should be taken into consideration [39]. Re-do CEA and CAS seems to be the same effect for the prevention of ipsilateral stroke [78, 79], but its choice must depend upon pathophysiology of stenosis mentioned above. If the restenosis occurs in early phase (<2 years) and intimal hyperplasia is suspected with plaque imaging, CAS has a priority because plaque rupture is hard to occur during stenting procedure. On the other hand, CEA with patch angioplasty may be better to the lesion which has been caused more than 3 years after initial CEA and has a sign of atheromatous plaque in the echo or MRI imaging.

10.11 Postoperative Management

The patients are recovered from anesthesia soon after surgery, but those who have been treated with dual antiplatelet therapy (DAPT) are kept anesthetized overnight to prevent postoperative bleeding. Patients are strictly monitored in the intensive care unit (ICU) or rooms comparable to ICU. Postoperative airway obstruction due to a carotid rupture or wound hematoma can occur mainly within 24 h postoperatively, which sometimes becomes fatal. If it occurs, emergency wound reopening and decompression should be performed. Blood pressure is kept under 80–100% of preoperative value until the SPECT denies postoperative hyperperfusion. If the hyperperfusion is recognized in SPECT, strict control of blood pressure should be continued for at least 4–7 days even if it is asymptomatic, because it can cause massive and sometimes fatal intracranial hemorrhage. Single antiplatelet therapy (SAPT) is restarted soon after surgery and continues thereafter [80–82].

10.12 Complication and its Management

10.12.1 Myocardial Infarction

This is most common systemic complication in CEA. As mentioned before, preoperative screening is essential to avoid this complication, but if preoperative coronary evaluation was insufficient, electrocardiogram (ECG) monitoring should be continued for a few days postoperatively. It is important to recognize that carotid artery stenosis is a part of systemic vascular disease, and vascular surgeons must keep in touch with cardiologists.

10.12.2 Nerve Palsies

Hypoglossal nerve and superior laryngeal nerve palsy can be occurred in CEA. The latter one, especially, can cause hoarseness and dysphagia and affect the quality of life. Most of the symptoms will recover within 3 months, but not completely in some patients. To avoid superior laryngeal nerve palsy, care should be taken not to dissect the tissue around ECA and superior thyroid artery too much, because this nerve usually runs just behind these arteries.

10.12.3 Cerebral Hyperperfusion Syndrome (CHS)

CHS has been reported in 0.2–18.9% of cases following CEA, but recent report showed less incidence (1.9%) [83]. It is well-known that the patients whose cerebral blood flow was severely decreased before surgery have dysregulation of cerebral vascular system and have a tendency of CHS [49]. The major symptoms of CHS include headache, restless, and seizure that appear in parallel with blood pressure elevation [48]. It is also reported that intracerebral hemorrhage (ICH) can be caused by CHS, and this ICH sometimes becomes fatal even though the incidence is quite low (0.37%) [83]. This complication is preventable, so screening the patient who is prone to CHS and postoperative BP control (<100% of preoperative value) with CBF evaluation (SPECT) are essential.

10.13 CEA for High Risk Patients (Advanced)

High risk for CEA is defined in Table 10.2. Comorbidities listed in this table are the risks for general anesthesia and if they are poorly controlled, CEA with general anesthesia becomes contraindication. As for risks for anatomical factors, most of

Table 10.2 High risk for CEA

Anatomical factors	Comorbidities
Previous neck surgery or tracheostomy	Severe CHF
Restenosis after CEA	Severe CAD
Previous radiation therapy	Severe pulmonary disease
Contralateral carotid occlusion	CKD
Contralateral laryngeal nerve palsy[a]	
High carotid bifurcation (above C2 vertebra)	

CHF chronic heart failure, *CAD* coronary artery disease, *CKD* chronic renal failure
[a]True contraindication for CEA among anatomical factors (personal opinion)

them can be overcome and not contraindication. For example, it has been reported that CEA for carotid stenosis with previous radiation therapy has longer stroke prevention with less restenosis than CAS, whereas cranial nerve palsy was more common [84–86]. CEA for contralateral carotid occlusion (CCO) seems not to be contraindication, because some reports demonstrated that perioperative stroke risk was not different between CCO and non-CCO patients, under routine or selective shunt [87, 88]. In our institute, among anatomical factors listed in Table 10.2, only contralateral laryngeal nerve palsy is thought to be contraindication of CEA. In case of previous neck surgery or tracheostomy, we usually use microscope to do meticulous dissection around carotid artery when adventitia and surrounding tissues are tightly adhered, although all of these patients have not been treated only with CEA.

10.14 Summary

CEA is a surgery for stroke prevention, whose efficacy is supported by many RCTs and whose recommendation level is quite high. Even though the devices and techniques of CAS progress, CEA seems to be golden standard for the intervention for carotid stenosis by its curability. To warrant its superiority, low complication rate is required, so CEA surgeons should continue to brush up their knowledge and skills. On the other hand, CEA, CAS and medical therapy are no longer competitive, but complementary treatment, so vascular surgeons should also catch up the current status of other two options and become able to change their surgical indication flexibly to give an optimal treatment to the patients.

Conflict of Interest Authors do not have any conflict of interest in this manuscript.

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