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Society for Vascular Nursing

Carotid Endarterectomy (CEA) Updated Nursing Clinical Practice Guideline

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1. Introduction

1.1.Purpose and Scope:

The purpose and scope of this document is to update the previously-published Society for Vascular Nursing's (SVN) 2009 carotid endarterectomy (CEA) practice guideline to reflect the evidence-based changes. Nursing practice in the care of the patient undergoing carotid artery stenting are detailed in the 2013 SVN guidelines.[1]

1.2. Assessment of Scientific Evidence

The comprehensive search strategy identified studies published in the English language from 1990-2015. Earlier studies were included if they remained clinically relevant. Search terms used were: Carotid endarterectomy, carotid artery surgery, extracranial vascular surgery and combinations thereof. The databases searched included: CINAHL, The Cochrane Library, Elsevier Science Direct, Ovid, MEDLINE, PubMed, BMJ Clinical Evidence, EBSCO, National Guidelines Clearinghouse, MD Consult, Nursing Consult and TRIP (Turning Research into Practice). Textbooks and review articles were also included. Recommendations for nursing practice are based upon data classified using a data grading system proposed by Melnyk and Finout-Overholt [2] and modified from the American Association of Neuroscience Nurses.[3]

The SVN clinical practice guideline classifies the data as follows:

1. Class I: Randomized control trial without significant limitations, systematic reviews or meta-analysis
2. Class II: Randomized control trial with important limitations (e.g., methodological flaws or inconsistent results), observational studies (e.g., cohort or case-control)
3. Class III: Qualitative studies, case study, or series
4. Class IV: Evidence from reports of expert committees and/or expert opinion of the guideline panel, standards of care and clinical protocols, animal studies

1.3. History of Carotid Endarterectomy

Gowers in 1875 is the first individual recognized with associating extracranial atherosclerosis with stroke.[4] The first successful carotid operation reported in the medical literature was performed by Felix Eastcott in 1954 in London that involved a carotid bifurcation lesion resection and primary anastomosis.(Eastcott, Pickering et al. 1954) Although not reported until 1975, DeBakey in 1953 performed the first successful CEA in the United States.[5] CEA is most frequently performed to prevent stroke. In 2010, an estimated 100,000 inpatient CEA procedures were done in the United States[6] with an incidence of 2.6 per 1000 patients per year.[7]

1.4. Rationale for Guideline:

Stroke is the fifth leading cause of death in the U.S. and the leading cause of serious long-term disability.[6] Between 2012 and 2030, total direct stroke-related costs are projected to triple, from $71.6 billion to $184.1 billion, with the majority of the projected increase in costs arising from those 65 to 79 years of age.[8] This represents a significant source of morbidity and mortality. Atherosclerosis is the cause of one-third of all strokes and approximately 50% of strokes occur in the distribution of the carotid arteries. Carotid revascularization is the current standard of care to prevent stroke in asymptomatic patients

with moderate to severe carotid artery stenosis especially if the perioperative risk of stroke, myocardial infarction (MI) and death are low.[9] Carotid revascularization is also recommended for patients with a recent transient ischemic attack or stroke due to carotid atherosclerosis, as carotid revascularization reduces the risk of recurrent neurologic events.

1.5. Clinical Practice Guideline Goal

This evidence-based clinical practice guideline was developed by vascular clinical nurse experts who are members of the SVN Practice and Research Committee to assist the registered nurse in delivering the optimal evidence-based care for the patient undergoing CEA. This document should be reviewed prior to implementation and tailored for each facility based on the needs of the practice setting and the values and preferences of the patient. A summary abbreviated checklist of nursing activities in caring for a CEA patient is included in Appendix A.

The goals of caring for this patient population are to:

1. Provide optimal nursing care based on clinically recommended practice guidelines.
2. Provide a safe and caring environment throughout each phase of the patient's experience before, during, and after carotid artery endarterectomy.
3. Effectively assess, plan, implement, and evaluate individualized patient care.

1.6 Review of Carotid Artery Anatomy and Physiology

There are two common carotid arteries**.** They differ in length and in their origin. The right common carotid artery begins at the bifurcation of the innominate artery behind the sternoclavicular joint and is confined to the neck. The left common carotid artery originates from the aortic arch to the left of the innominate artery.[10] The thoracic portion of the left common carotid artery ascends from the arch of the aorta through the superior mediastinum to the level of the left sternoclavicular joint, where it is continuous with the cervical portion. In the neck, each common carotid artery is contained in a fascia sheath that also encloses the internal jugular vein and vagus nerve. As the common carotid artery passes upward in the neck to the upper border of the thyroid cartilage, it divides into the external and internal carotid arteries.[10]

The internal carotid artery begins at the bifurcation of the common carotid artery typically opposite the upper border of the thyroid cartilage. The internal carotid artery commonly runs vertically upward in front of the upper three cervical vertebrae through the carotid canal winding through the dura forming the carotid siphon before reaching the brain.[11] It supplies blood to the inside of the head: the anterior part of the brain, the eye and its appendages, and sends branches to the forehead and nose. The carotid anatomy is depicted in Figure 1. Just above the carotid bifurcation at the origin of the internal carotid artery is a localized slightly widened area known as the carotid sinus or "bulb". The carotid sinus contains a large number of baroreceptors, which are specialized nerve endings within the sinus wall adventitia.[12] These receptors (also present in a similar amount in the aorta and smaller numbers in other large arteries) function on a negative feedback system with the autonomic nervous system to provide short-term control of arterial pressure.[12] Simplistically, the baroreceptors are stretch-type mechanical receptors that send impulses from the carotid sinus up the glossopharyngeal nerve to the medulla in response to

pressure-related changes in the vessel size. The higher the pressure, the more frequently the baroreceptors fire. This results in the medulla inhibiting the sympathetic nerve impulses to the peripheral vessels, thus lowering the blood pressure.[13]

The external carotid artery is more superficial and also typically begins at the level of the upper border of the thyroid cartilage. It takes a slightly curved course and passes upward and backward in the neck. The external carotid artery rapidly becomes smaller as it divides into four sets of branches: anterior, posterior, ascending and terminal.[10] It advances up the neck within the parotid gland to the space behind the mandible. It supplies blood to exterior head; specifically the face and scalp.

1.7 Pathogenesis of Carotid Atherosclerotic Disease

Stroke is a leading cause of death and disability worldwide. Approximately 87% of strokes have an ischemic origin.[6] In 2014, strokes were responsible for 1 in every 20 deaths in the United States.[6] One-third of all ischemic strokes are felt to be secondary to emboli. In patients without atrial fibrillation, approximately 90% of these emboli are atherogenic in nature and originate extracranially from the proximal internal carotid artery.[14] The risk of stroke is dependent upon the degree of carotid artery stenosis. In patients with 75% or less narrowing, the stroke incidence is approximately 1.3% per year. If the stenosis is 75% or greater in the symptomatic patient, the stroke rate rises to about 10.5% per year.[9] The risk of stroke in the asymptomatic patient is much lower, approximately 1-2.4% per year.[15]

Atherosclerosis and inflammation have a similar mechanism during the early phases of plaque development, which involves increased interaction between the vascular endothelium and leukocytes.[16] The earliest lesion of atherosclerosis is known as the fatty streak, which is an infiltration of monocyte-derived macrophages and T-lymphocytes into the arterial intima. The fatty streak starts as an infiltration of low-density lipoprotein (LDL) cholesterol in the arterial wall, followed by its oxidation.[16] Over time the fatty streaks enlarge and combine to form plaques. The process continues when macrophages secrete multiple biochemical mediators that induce inflammation and smooth muscle cell proliferation within the arterial wall surrounding the plaque.[16] The lipids and cells can enlarge into the arterial lumen resulting in diminished blood flow.

Atherosclerosis is most severe in the posterior wall of the carotid sinus, where there is low shear stress and greater turbulence.[17] This leads to cumulative vessel wall metabolic disturbances, prolonged exposure to plasma lipids and release of other inflammatory mediators. The developing plaque extends downward at the bifurcation into the common carotid artery (Figure 2). Most myocardial infarctions have been associated with thrombosis in plaques with high inflammatory cell content and large necrotic lipid cores, the so-called unstable plaques. Symptomatic carotid artery plaque pathology is similar to coronary artery plaque.[18] Symptomatic carotid artery disease is defined as a sudden onset of focal neurologic symptoms that are attributable to emboli from the narrowing of the carotid artery. Symptoms can include one or more transient ischemic attacks characterized by temporary focal neurologic dysfunction, transient monocular blindness (amaurosis fugax), or one or more non-disabling ischemic strokes.[19] Risk factors for carotid

atherosclerosis are similar to coronary artery disease including cigarette smoking, diabetes, race, age, obesity and hypertension.[20]

1.8 Selection Criteria for Carotid Endarterectomy and Summary of Evidence

Surgical selection criteria are based upon three clinical trials below:

a. North American Symptomatic Carotid Endarterectomy Trial (NASCET) – original and surgical arm results
b. European Carotid Surgery Trial (ECST) original and reanalysis.
c. Carotid Endarterectomy and Prevention of Cerebral Ischemia in Symptomatic Carotid Stenosis Study.

Based on the results of these studies, the American Heart Association concluded that carotid endarterectomy is beneficial for symptomatic patients with a recent non-disabling neurological event and ipsilateral stenosis of 50-99%; and it has uncertain benefit for symptomatic patients with < 50% stenosis. Carotid endarterectomy reduced the risk of disabling stroke or death for patients with stenosis exceeding that measured in ECST (70%) or NASCET (50%). This result applies only to surgically fit patients operated on by surgeons with low complication rates. In asymptomatic patients with carotid stenosis, clinical trial data suggest that the degree of stroke prevention from CEA is less than among symptomatic patients.

The North American Symptomatic Carotid Endarterectomy Trial (NASCET)[21] randomized patients with carotid artery stenosis of 70% to 99% who had transient hemispheric symptoms, amaurosis fugax, or a non-disabling stroke to carotid endarterectomy or optimal medical care, including antiplatelet therapy.[22] The cumulative risk of any ipsilateral stroke at 2 years was 26% in the medical care group and 9% in the surgical group[22] (Class I). This resulted in an absolute risk reduction of 17%. Carotid endarterectomy was also found to be beneficial when all strokes and deaths were included in the analysis [22] (Class I). A further analysis of the 1415 patients who underwent a CEA in the surgical arm of NASCET found 2% overall risk of permanently disabling stoke and death.[23]

The European Carotid Surgery Trial (ECST) showed an 11.6% benefit (determined by Kaplan-Meier estimate) from CEA after 3 years in patients with stenosis > 80% in diameter. The Asymptomatic Carotid Surgery Trial (ACST) demonstrated a relative risk reduction of 46% in patients who have 60-99% asymptomatic carotid stenosis who underwent CEA. An estimated yearly stroke rate of 2% in patients treated medically can be reduced to 1% with CEA in asymptomatic patients. [24] A reanalysis of the ECST results found that surgery was highly beneficial in patients with 70% to 99% stenosis and moderately beneficial for 50% to 69% stenosis [25] (Class I).

The CEA benefits on asymptomatic patients with high-grade stenosis are not as robust and are based upon the following three trials and meta-analysis.

a. Veterans Affairs Cooperative Study Group
b. Asymptomatic Carotid Atherosclerosis Study (ACAS)
c. Asymptomatic Carotid Stenosis Trial (ACST)

The Veterans Affairs Cooperative Study Group trial[26] was a multicenter, randomized clinical trial of 444 asymptomatic men with 50% or greater carotid artery stenosis by angiography. Subjects were randomly assigned to optimal medical treatment including aspirin plus CEA (211 patients) or aspirin alone (233 patients). The combined incidence of ipsilateral stroke was 8% in the CEA group and 20.6% in the medical group. There was no difference in the 30-day stroke and death rates between the two groups.[26]

The Asymptomatic Carotid Atherosclerosis Study (ACAS) was a randomized clinical trial assessing the efficacy of treating asymptomatic carotid stenosis with low dose aspirin (300 mg/day) alone versus carotid endarterectomy in conjunction with the daily administration of low dose aspirin. [27] More than 42,000 patients were screened. Of those screened, 1,662 asymptomatic patients were randomized to CEA plus medical therapy (n=825) or medical therapy alone (n=834). The 30 day perioperative stroke or death rate was 2.3%, with an additional 1.2% stroke incidence due to carotid angiography. Carotid endarterectomy significantly cut the 5 year risk of ipsilateral stroke, perioperative stroke, or death in half (from 11% to 5.1%) [9, 27, 28] (Class I).

The Asymptomatic Carotid Stenosis Trial (ACST) randomized 3,120 asymptomatic patients with $\geq$ 60% carotid artery stenosis by ultrasound.[29] There was a 2.8% risk of stroke or death within 30 days for those randomized to CEA. The 5 year risk of stroke was reduced significantly for CEA (6.4%) compared with medical therapy (11.8%). At 5 years there was no difference in the rates of all stroke and/or death between medical therapy and CEA.[29]

A meta-analysis by Chambers and Donnan was conducted on the above three asymptomatic CEA trials that included a total of 5,223 patients. It was noted that the risk of ipsilateral stroke or any stroke was reduced by approximately 30% over three years. However, the absolute risk reduction was small (approximately 1% per annum over the first few years of follow up.[30, 31] The authors speculated that the risk reduction may be higher with a longer follow up. Currently, asymptomatic patients with a high-grade carotid stenosis, reasonable life expectancy and perioperative risk less than 3% may be considered candidates for a CEA[32] (Class I).

Guidelines published in 2011 by Ricotta, et.al. from The Society for Vascular Surgery[33] included the following summary recommendations for carotid surgical revascularization:

- Asymptomatic patients with $\geq$ 60% stenosis should be considered for CEA for reduction of long-term risk of stroke provided there is a 3-5 year life expectancy and perioperative stroke/death rates are less than 3%
- Patients >70 years of age, with long (>15-mm) lesions, pre-occlusive stenosis, or lipid-rich plaques that can be completely removed safely by a cervical incision in patients who have a virgin, nonradiated neck

Encouraging research that targets the impact of optimal medical management alone on reducing carotid plaque progression and stroke in patients with symptomatic and asymptomatic carotid stenosis is being advocated.[34] Optimal medical treatment includes

aggressive risk factor management such as smoking cessation, blood pressure reduction, regular exercise, glycemic control, and lipid lowering, among others. The forthcoming Carotid Revascularization Endarterectomy Versus Stenting Trial 2 (CREST-2) is testing a hypothesis that intensive medical treatment (IMT) alone is superior to an invasive carotid intervention, (CEA or carotid stent) plus IMT in the prevention of stroke and death.[35] Twelve hundred and forty patients will be randomized to CEA plus IMT versus IMT alone and an additional 1240 patients will be randomized to carotid stent plus IMT versus IMT alone.[35] The results of this trial may modify the selection criteria for CEA among asymptomatic patients.

1.9 Simplified Procedural Technique for Carotid Endartarectomy

1.9.1 A carotid endarterectomy is performed in an operating room under sterile technique. There are several types of surgeons that perform CEA's including vascular, general, cardiothoracic and neurosurgeons.

1.9.2 Anesthesia types may include local, regional, superficial cervical block or general anesthesia as determined by physician preference and patient co-morbidities [36] (Class I).

1.9.3 The patient is positioned supine. A blanket roll may be placed under the scapula to allow for some neck hyperextension. Once anesthesia is achieved, the table is placed in a reverse Trendelenberg position.[37]

1.9.4 The typical skin incision may either be longitudinal or transverse (Figure 3). The standard incision is longitudinal which is made parallel to the medial border of the sternocleidomastoid muscle. The transverse incision is made in the skin crease, approximately 1 to 2 cm inferior to the angle of the jaw.[37]

1.9.5 The incision is carried through the platysma. Retraction of the sternocleidomastoid muscle is done to expose the carotid sheath and internal jugular vein.

1.9.6 Further dissection occurs. The internal carotid artery (ICA) is identified and clamped beyond the distal aspect of the plaque. The common carotid and external carotid arteries are clamped. Heparin is administered (70-100 units/kg) to maintain an activated clotting time (ACT) between 200-250 seconds.[37]

1.9.7 The surgeon determines the need for a shunt. The use of a silicon tube, or shunt, as a temporary bypass has been postulated to reduce the length of time that blood flow to the brain is interrupted during the operation. This may reduce the risk of a perioperative stroke.[37] The type of shunt used is dependent upon physician preference. There is limited data to either support or negate routine shunting in a CEA[38] (Class I). Other methods of cerebral monitoring include carotid stump pressure, electroencephalography and cerebral oxygen saturation.[37]

1.9.8 The conventional method for plaque extraction is for a longitudinal incision made in the ICA. The atherosclerotic plaque is removed from the vessel by pulling the

plaque out transversely or by transecting with scissors. Attention is then paid to any residual debris and the end points of the endarterectomy. Tacking stitches may be placed. [37]

1.9.9 An alternative method of performing a CEA is known as eversion endarterectomy. The exposure of the vessel follows the same technique as described above. A test clamp of the ICA is done to assess for cerebral ischemia before proceeding. The rationale for the test clamp is that it is difficult to shunt if using this technique. If no cerebral ischemia occurs, the ICA is transected at its origin and turned inside out until the distal plaque is exposed and then it is transected. The ICA is re-anastomosed to the carotid bifurcation.[37] Eversion endarterectomies do not require a patch closure. Originally felt to be associated with a lower incidence of perioperative stroke and restenosis, current evidence does not support these assumptions and the choice of endarterectomy technique should be based upon the expertise of the surgeon[39] (Class I).

1.9.10 ICA arteriotomy closure can be primary using nonabsorbing sutures or include a patch (known as patch angioplasty) (Figure 4). Patch materials include autologous vein, woven polyester (Dacron), polytetrafluoroethylene (PTFE) and bovine pericardium. Limited evidence suggests the use of patch angioplasty during arteriotomy closure may reduce the incidence of restenosis[38](Class I). The Society for Vascular Surgery recommends patch closure or eversion endarterectomy over primary closure because of the risk of restenosis[33](Class I). There is some evidence that synthetic (PTFE) patches may be superior to collagen impregnated Dacron grafts in terms of perioperative stroke rates and restenosis[40] (Class I). Pseudoaneurysm formation may be more common after use of a vein patch compared with a synthetic patch.[37, 41]

1.9.11 Flushing is performed, and if used, the shunt is removed before final artery closure. Use of an arterial Doppler duplex scanning device or intraoperative angiography may be used to assess for artery patency.[37, 41]

1.9.12 Generally, the underlying tissues are closed with absorbable sutures and the skin incision is closed with a subcuticular suture Heparin reversal with protamine sulfate is determined per physician preference and patient co-morbidities.[42, 43] Administration of intraoperative intravenous dextran is another method sometimes used to reduce the risk of stroke as it has antithrombotic properties; however, its use has been associated with a higher incidence of myocardial infarction and heart failure.[44] Topical hemostatic agents may also be used to achieve surgical hemostasis. Insertion of a vacuum bulb-type suction drain into the incision may be done at this time. The choice of external dressing is dependent upon physician preference.

2. Pre-Operative Nursing Care

2.1 Nursing Assessments

2.1.1 Assess for co-morbid conditions and risk factors for atherosclerosis. Risk factors for carotid atherosclerosis are similar to coronary artery disease including cigarette smoking, diabetes, race, age, obesity and hypertension.[633] Evaluate for the presence of other neurological diseases that may impact the postoperative course such as Alzheimer's, epilepsy or Parkinson's.[45] Ascertain if there is a history of previous carotid revascularization, neck surgery or a history of neck radiation.

2.1.2 Obtain a list of current medications that the patient is taking including over-the-counter drugs. Assess for allergies or hypersensitivities to medications such as IV contrast, foods or latex.[46-48]

2.1.3 Obtain baseline vital signs to include apical/radial pulse, respirations, pulse oximetry, temperature and blood pressure. Use a standard technique for obtaining the blood pressure including the appropriate cuff size and supporting the arm at heart level[49] (Class IV). Include bilateral upper extremity blood pressures. Unequal upper extremity blood pressures greater than 15mmHg difference in systolic blood pressure (SBP) may indicate subclavian artery stenosis.[50] Inform physician if this SBP difference is noted. Notify the surgeon and anesthesiologist if the systolic blood pressure is greater than 180mmHg or diastolic blood pressure is greater than 110mmHg within two (2) hours of the surgery start time. The surgeon may consider postponement due to an increased risk of stroke from these pressures or place the patient on additional antihypertensive medications.[51, 52]

2.1.4 Perform a targeted physical assessment. Notify the physician of any abnormal findings. Excluding the neurological assessment, there is a lack of evidence regarding the precise assessments that are required; however clinical practice recommendations include: general appearance, status of the skin (integrity, presence of lesions/edema, nails), head/neck, chest including breath sounds/heart sounds, abdominal assessment (distention/bowel sounds/genitals), bilateral extremity motor strength and movement, temperature, color, sensation, pulses and capillary refill.[53] Grade the pulses according to institutional protocol.

2.1.5 Neurological Assessment to include a focused neurological examination including level of consciousness, speech pattern, pupils, facial symmetry, gait, muscle atrophy, fasciculations or involuntary movements and cranial nerves. [54] Consider performing a Glasgow Coma Scale, and consider a baseline National Institutes of Health Stroke Scale (NIHSS) or other stroke scale assessment. These provide documentation of any pre-interventional neurological deficits. Refer to Appendix B for detailed description of pertinent cranial nerve assessment. The National Institute of Health Stroke Scale NIHSS[55] is a tool for assessment of neurological impairment associated with stroke reflecting global and/or focal changes in brain vascular territories. Neurological assessment should be performed within 24 hours of surgery[56] (Class IV).

2.2 Carotid Diagnostic Studies

Confirm completion of baseline carotid artery testing which has confirmed location and degree of stenosis[15] (Class I). The choice of test is dependent upon the individual physician. Options include carotid arterial Doppler and/or duplex, transcranial Doppler, magnetic resonance angiography or computed tomography angiography. These diagnostics provide an assessment of the lesion characteristics.

2.2.1 Carotid Duplex Ultrasound (CDUS) is a non-invasive study utilizing high-frequency sound waves to produce pictures inside the carotid and vertebral arteries. Doppler waveforms and color flow imaging can identify direction and velocity of blood flow in the carotid and vertebral arteries. Interpretation of velocities should be done by qualified and experienced care providers. This procedure can be done as an outpatient. There is no patient preparation except to instruct patients to wear comfortable, loose-fitting clothing and no jewelry over the area to be scanned. Advantages of CDUS are that it provides accurate and reliable information, and is noninvasive, but accuracy is dependent on the sonographer's technical skills. Velocities can be affected by contralateral carotid artery occlusions and by gender; women tend to have higher velocities [57, 58] The presence of carotid stents,[59] obesity, tortuosity of arteries, high carotid bifurcation and severe intravascular calcification can reduce the reliability of CDUS.[33] Table 1 summarizes one example of a consensus of the velocity criteria for internal artery carotid artery stenosis. Interpretation of velocities should be done by qualified and experienced care providers in an accredited lab.

Table 1: Doppler Ultrasound Velocity Criteria of Internal Carotid Artery Stenosis

Degree of Stenosis	Peak systolic velocity (cm/sec)	End diastolic velocity (cm/sec)	Systolic velocity ratio (ICA/CCA)
<50%	<150	<50	<1.8
50-69%	150-250	50-90	1.8-2.8
70-89%	>250-400	>90-150	>2.8-5.0
90-99%	>400	>150	>5.0

Adapted from: Grant EG et al. Carotid artery stenosis: Greyscale and Doppler ultrasound diagnosis. Society of Radiologists in US consensus conference. Radiology 2003; 229: 340-6.
ICA: Internal carotid artery; CCA: common carotid artery

2.2.2 Magnetic Resonance Angiogram (MRA) uses radio waves and magnetic fields to create detailed images showing blood flow inside the vessels. Gadolinium, a non-iodinated contrast media, may be used to improve the test's accuracy by making the arteries more visible. Gadolinium is not without consequence as it can trigger nephrogenic systemic fibrosis in those with a history of renal disorders.[60] MRA can precisely define the dimensions and extent of aortic arch aneurysms, dissections, vascular tumors and periaortic abscesses comparable to transesophageal echocardiogram (TEE). It also can identify structure of atherosclerotic plaque.[61] It can be used to monitor carotid and vertebral stenosis. Patient preparation for MRA must include history of contrast allergies and any metal implants in their body. These include pacemakers or implantable cardioverter defibrillator (ICD); surgical metal rods, screws, plates, stents, clips,

pins, staples and wires (some may not be exclusionary), intravascular embolization coils, cochlear implants, metal heart valves, shrapnel or bullets, implantable ports and nerve stimulators. Removable dental work and body piercings must be removed. Advantages of MRA are that it is noninvasive, with no ionizing radiation or iodinated contrast and it presents numerous projections of the carotid lumen from a single test. MRA can visualize arteries beyond coverage area for CDUS. Disadvantages include the possible over-estimation of the degree of stenosis and that it is difficult to perform in claustrophobic or uncooperative patients.[62]

2.2.3 Computed Tomography Angiogram (CTA) utilizes non-contrast technique or IV contrast to identify the specific anatomy of the aortic arch, carotid arteries, and intracerebral arteries. CTA uses ionizing radiation when taking x-ray pictures in the form of slices of the brain, carotid and vertebral arteries, showing areas of poor blood flow or the presence of cerebral aneurysms. Contrast media may be injected to enable visualization of blood vessels on x-ray to identify area(s) of arterial stenosis. The patient should be instructed to have nothing by mouth (NPO) at least four hours before the scheduled exam. A thorough history of allergies and specific types of reaction to previous contrast media should be completed. Baseline renal function tests of BUN and creatinine are completed to ensure safety of contrast administration. Premedication and IV hydration may be given in the case of contrast allergy or renal insufficiency. Metformin should be held as noted below. The patient should wear comfortable clothing but may be asked to change into a gown and remove hairpins, hearing aids, glasses, metal dental work, necklaces, earrings and any body piercings on the head. Advantages of CTA are that it is less likely to over-estimate the degree of carotid artery stenosis, high-quality spiral CT images can be evaluated in multiple planes, CTA is less expensive and faster than MRA, multiple organs can be visualized simultaneously, and a wide anatomical range is viewed from the aortic arch to circle of Willis. Disadvantages are that it is more expensive than CDUS, there is contrast and ionizing radiation exposure, and intravascular calcium burden can affect visualization, and assessment of plaque structure.[63] The decision to use either CTA or MRA is location-specific due to operator proficiency and the availability of the appropriate equipment.

2.2.4 Carotid Artery Contrast Angiography has previously been considered to be the gold standard for diagnosing the degree and location of carotid stenosis. This test is generally performed if discrepancies exist between other diagnostic modalities such as CDUS and MRA, especially in renal-impaired patients. Its current role in extracranial vascular disease is mainly limited to carotid artery stenting. The NASCET method is generally used for interpretation of results.[61] The NASCET method uses the diameter of the proximal internal carotid artery above the carotid bulb as the reference diameter.[14] The patient should be instructed to have nothing to eat or drink for six to eight hours before the scheduled procedure. The patient will need to receive instruction on taking cardiac or other medicines the morning of procedure with sips of water. Instructions will be given regarding holding metformin 24 hours before exam and usually 24-48 hours after exam or until renal function returns to baseline (see preoperative medications). A thorough a history

and physical should be completed, including assessment for a history of allergies or reactions especially to contrast media. Baseline blood tests to check renal function, BUN and creatinine should be completed. Coagulation tests may be performed, PT/INR, PTT, platelet count, hemoglobin and hematocrit. A peripheral IV line will be started for hydration and to administer any renal-protective medicines as indicated. The advantages of angiography are that it can be used if patients have metal implants and cannot have other forms of diagnosing imaging, if patient is obese, and the ability to limit contrast burden in patients with renal impairment.[64] The incidence of neurological complications is low; approximately 1.3%, but in patients with cardiovascular disease, the neurological complication rate is higher at 2.3%.[65]

2.3 Diagnostic Laboratory Testing

2.3.1 Verify ordered pre-procedural laboratory tests have been done within the pre-established time period (usually within 7 days of the procedure or as specified by the institution). General recommendations are described by Iyer, et.al [66] and Fleisher, et.al.[67] Typical baseline laboratory tests include a complete blood count, blood urea nitrogen, serum creatinine, electrolytes, partial thromboplastin time, prothrombin time with INR, creatinine kinase and creatinine kinase MB fraction and urinalysis.[67,68] Include measurement of blood glucose in diabetic patients.

2.3.2 A 12-lead ECG is standard.[67]

2.3.3 Additional ancillary diagnostic ancillary studies may be ordered based upon the institutional protocol as well as patient age, gender, presence of severe obesity or comorbidities (e.g. pregnancy test, pulmonary function studies, stress test, chest x-ray).[67, 69] Notify physician of any abnormal results.

2.4 Anesthesia Evaluation

Confirm an anesthesia assessment has been scheduled before the surgery**.** A pre-surgical clinical assessment and evaluation by anesthesia accomplishes several goals including to plan for the selection of anesthetics, evaluate for a familial history of malignant hyperthermia and to assess the patient's airway. It also includes opportunities for patient education and allows the formation of a plan-of-care for the perioperative and recovery periods.[70] (Class I) CEA can be performed under general anesthesia or regional anesthesia; rarely CEA is performed under local anesthesia.[51, 71]

2.5 **Preoperative Patient Preparation/Education**

2.5.1 Clip any neck hair on a male patient at the selected surgical site with electric clippers as ordered[72, 73] (Class I). Clipping instead of shaving reduces the incidence of procedural/surgical site infections.[72, 73]

2.5.2 Cleanse patient preoperatively using a chlorhexidine shower or disposable wipes to reduce bacterial skin flora and the risk of surgical site infection.[74]

2.5.3 Establish peripheral IV access; depending upon institutional protocol, two IV sites may be preferred. Hypnotic agents and other medications are administered intravenously during the surgery.

2.5.4 Maintain nothing by mouth (NPO) for at least 8-hours before the procedure or as specified by the surgeon[75] (Class IV). NPO status reduces the potential for aspiration due to anesthesia.[75]

2.5.5 Assess the patient's neck mobility. Some neck flexibility is critical for proper surgical access and positioning.[51]

2.5.6 Provide additional patient education regarding medications, risk factor modification, peri-procedural and post-procedural care based upon institutional protocol. This education may include perioperative sequence of events, possible sensations (e.g., pain and anxiety), planned anesthetic mode, monitoring equipment, neurologic and vital sign checks, recovery progression and normal side effects.[76, 77] Blackburn and Neaton also recommend inclusion of discharge criteria and anticipated day of discharge in the pre-surgical education.[76]

2.6 Pre-Operative Management of Medications

Continue most medications that have withdrawal potential and hold non-essential medications the day of CEA surgery.[78] The reader is referred to additional sources for a broader overview of chronic medications in the preoperative setting.[51, 79]

2.6.1 Antiplatelet Agents

Aspirin 81 to 325mg daily is recommended before CEA[33, 51, 78, 80] (Class I). Administration of other antiplatelet agents such as clopidogrel, prasugrel or ticagrelor should be evaluated on a case-by-case basis with emphasis on the indication for which these agents were prescribed. The multidisciplinary team members involved in the decision should include the surgeon, cardiologist, anesthesiologist, primary care provider and patient.[81]

When an antiplatelet agent is prescribed along with aspirin, it is known as dual antiplatelet therapy (DAPT). If DAPT is prescribed for a cardiac stent patient, the recommendation is to continue DAPT for at least 4-6 weeks following placement of a bare metal stent (BMS) and at least 12 months after a drug eluting stent (DES).[81, 82] Stopping DAPT prematurely may place a patient at risk for stent thrombosis, myocardial infarction and death. An elective CEA procedure should not be performed during these time frames should DAPT cessation be advised in a particular case.[67]

Continued use of DAPT in persons undergoing non-cardiac surgery places them at risk for perioperative bleeding with a potential need for transfusions.[33, 82] However, CEA surgery (unlike open thoracic and abdominal aorta surgery) is not a procedure that is associated with a high risk for bleeding.[82] Several studies have shown no significant increase in perioperative bleeding in patients undergoing CEA while

taking clopidogrel, with or without aspirin. [83,84] Wait [84] maintains the risk of reoperation for neck hematoma evacuation is preferable to a perioperative stroke or MI should clopidogrel be stopped in the high risk patient.

2.6.2 Lipid-Lowering Therapy

Continue statin (HMG COA Reductase Inhibitor) medications the day of surgery [33, 51, 67, 80] (Class IV). It is considered reasonable to initiate a statin before the CEA in statin-naïve patients. [33, 67, 80] Although statins are known for their cholesterol lowering effects, it is their theorized reduction in vascular inflammation, stabilization of plaque and neuroprotective effects that supports their preoperative administration.[85] A study by the Vascular Study Group of New England showed reduced 30 day mortality and 18% improvement in 5 year survival after various vascular surgeries when antiplatelet and statin medication were utilized preoperatively and at discharge.[86] Hold non-statin medications such as bile sequestrants (cholestyramine and colestipol) which may interfere with bowel absorption of perioperative medications.[87]

2.6.3 Antihypertensives

2.6.3.1 It is reasonable to achieve good preoperative control of arterial pressure in patients who have SBP (systolic blood pressure) consistently > 180 mm Hg and who do not have severe bilateral carotid stenosis or frequent neurologic events attributable to carotid disease.[88]

2.6.3.2 Administer previously prescribed calcium channel blockers, alpha agonists, vasodilators and nitrates the morning of the procedure with a sip of water.[51, 79]

2.6.3.3 Clarify surgeon preference regarding preoperative administration of angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARB) medications. Literature is conflicting regarding use in this setting. Several authors report cases of prolonged postoperative hypotension, likely a result of the compensatory activation of the renin-angiotensin system.[51, 79, 89] In contrast, others advocate administration of these medications the day of surgery.[78, 90]

2.6.3.4 Hold any diuretic medication the day of the surgery[67, 78] (Class II). Loop and thiazide diuretics can cause hypovolemia and hypokalemia and can intensify the known hypotensive effects of anesthesia induction.[51, 78]

2.6.3.5 If the patient is already receiving a beta-blocker, administer as previously prescribed[68] (Class IV).

2.6.4 Diabetic Medications

Withhold metformin and its derivatives 24-hours before anesthesia to avoid the potential for lactic acidosis. [79, 91] Other oral hypoglycemic agents are held the morning of the procedure. Any prescribed long-acting insulin is adjusted based

upon the institution's protocol. Supplemental short-acting insulin may be administered during this period to maintain a target glucose level between 140-180 mg/dl in the critically-ill patient[92] (Class II).

2.6.5 Chronic Pain Medications

Stop all non-steroidal anti-inflammatory medications at least 3 days prior to surgery or as directed by the physician.[78, 93] These medications reduce platelet aggregation and increase the risk of bleeding by a decreased production of thromboxane.[93] Continue chronic opioid medications day of surgery to reduce irritability, diaphoresis, insomnia, nausea and other symptoms of withdrawal.[51]

2.6.6 Pulmonary Medications

Continue pulmonary inhalers day of surgery to reduce the incidence of postoperative complications.[93, 94] Hold theophylline the evening before surgery as it may precipitate arrhythmias and interact with anesthetic medications.[51, 93] Glucocorticoids (used for pulmonary or other indications) are continued day of surgery as to avoid symptoms of adrenal insufficiency; consider stress (supplemental) dose steroid if a patient has received oral corticosteroids for 3 consecutive weeks or more.[51]

2.6.7 Management of Antithrombotic Medications and Dietary Supplements.

2.6.7.1 Withhold any non-bridged unfractionated heparin or low-molecular weight heparin in the hospitalized patient based upon the institution's protocol

2.6.7.2 Withhold Vitamin K antagonists (VKA's) such as warfarin for approximately five days before the procedure (Class IV).[95] The INR should be normal or near normal (less than 1.5). If bridging therapy is required in the chronic atrial fibrillation or mechanical heart valve patient at high risk for venous thromboembolism (VTE), administer a therapeutic dose of a low-molecular weight heparin (LMWH) such as enoxaparin. The last LMWH dose should be administered approximately 24 hours before the procedure (Class IV).[95] If the patient is receiving therapeutic-dosed unfractionated heparin (UFH) as the bridging medication, the last dose should be stopped within 4 to 6 hours of the procedure (Class IV).[95] In the chronic atrial fibrillation or mechanical heart valve patient who is at a low risk for VTE; no bridging therapy is recommended [95] (Class IV). Resume VKA's 12 to 24 hours after the procedure[95] (Class IV).

2.6.7.3 Withhold the oral antithrombotic agent dabigatran for 1 to 2 days before the procedure in patients with a creatinine clearance greater than or equal to 50ml/minute. If the creatinine clearance is less than 50ml/minute, withhold medication for 3 to 5 days. A longer withholding time may be considered in patients where total hemostasis is necessary.[96] If the patient is prescribed rivaroxaban, the drug is stopped at least 24 hours before the procedure. [97] Apixiban should be held at least 24 hours in elective procedures that are considered to be low-risk for clinically significant bleeding. If the

procedure is considered moderate to high risk for clinically significant bleeding; Apixiban should be withheld for 48 hours 2012).[96, 98] If the patient is on edoxaban, the drug should be withheld for 24 hours before the surgery.[99]

2.6.7.4 Instruct the patient not to take any herbal or dietary supplements for the specified number of days prior to the procedure, based upon the institution's protocol. There is an increased potential for bleeding and alteration in the effects of medications such as warfarin[100-103](Class IV). Ginkgo, Vitamin E and garlic (allium sativum) demonstrate antiplatelet activity and increase bleeding time[100-102] (Class I). Case reports have shown an interaction between herbal supplements St. John's Wort (hypericum perforatum) and American ginseng that decrease the INR[103](Class IV). Dansehen (salvia miltiorrrhiza) increases the INR[100] (Class IV).

3. Operative Care

3.1 Before Patient Enters the Operating Room

3.1.1 Verify correct patient identification, operative procedure and side of carotid operation; this is done every time the care responsibility of a patient is transferred to another person in the perioperative course to avoid wrong-site surgery utilizing a standardized pre-surgical checklist[104,105] (Class I).

3.1.2 Ascertain that the patient has signed the procedural consent and anesthesia consent. Confirm the surgeon has signed the procedural consent and the anesthesiologist or nurse anesthetist has signed the anesthesia consent.

3.1.3 The correct operative site is marked with a permanent marker by the surgeon or a member of the operative surgical team as determined by institutional protocol. This is best done with input from the awake and alert patient (or care giver) to prevent wrong-site surgery.[104,106]

3.2 Intra-Operative Care

3.2.1 Assist with attachment of ECG monitor leads, non-invasive blood pressure cuff and pulse oximetry probe to the patient once patient is on the operating room table. If indicated, obtain baseline blood pressure, heart rate, respirations, SpO_2 and ECG rhythm.

3.2.2 Assist with setup and insertion of an arterial line as directed. Carotid artery manipulation predisposes the patient to potential changes in perioperative blood pressure that may require vasoactive medications.[89] The use of continuous intra-arterial monitoring allows for earlier recognition and treatment of blood pressure fluctuations that may lead to intraoperative or postoperative neurological complications.

3.2.3 Position the patient's head and neck based upon physician preference and proposed anesthetic technique. This allows for patient comfort and sufficient site exposure that includes some neck hyperextension.[107] If the procedure is performed under general

anesthesia, the endotracheal tube is taped to the corner of the mouth on the opposite side of the surgical field.[107]

3.2.4 Administer the ordered intravenous (IV) antibiotic prophylaxis within one (1) hour of surgical incision at the recommended infusion rate to reduce the incidence of surgical site infection[108] (Class I).

3.2.5 If general anesthesia is utilized, observe for the development of malignant hyperthermia. Malignant hyperthermia (MH) is a genetic disorder that is precipitated by use of volatile anesthetic agents (halothane, enflurane, isoflurane, desflurane, and sevoflurane) or the muscle relaxant succinylcholine, or both.[109] It is characterized by life-threatening, extremely high fever (greater than 110 degrees Fahrenheit), skeletal muscle spasms, hypotension, change in level of consciousness, tachypnea and tachycardia. Acidosis, hyperkalemia, cardiac arrhythmias and rhabdomyolysis may occur.[110] Early recognition is integral to preventing mortality. Immediate treatment consists of switching to a non-inducing agent, dantrolene 2 mg/kg IV, repeated until the cardiovascular symptoms stabilize to a maximum of 10mg/kg[109, 110] (Class IV). In addition, hyperventilation at 100% oxygen, application of external cooling devices and administration of chilled IV normal saline are used (Class IV).[110]

3.2.6 Assist physician if indicated with placement of selected cerebral monitoring device. Cerebral monitoring during a CEA assists in reducing the perioperative stroke rate from embolism, hypoperfusion and postoperative hyperperfusion syndrome.[111,112] If used, the selection of a cerebral monitoring device is dependent upon physician preference and includes: electroencephalography, transcranial Doppler, stump pressure, sensory-evoked potentials and near-infrared spectroscopy.[111]

3.2.7 Monitor blood pressure and ECG rhythm as indicated with anesthesia. Notify anesthesia and the surgeon immediately if hypotension or hypertension occurs. Bradyarrhythmias are typically due to temporary baroreceptor dysfunction and treatment is generally not required.[41] Blood pressure fluctuations are due to multifactorial causes that may include: prior history of hypertension, carotid manipulation, pain-induced sympathetic nervous system stimulation and anesthesia induction.[52] Research suggests maintaining a mean arterial pressure (MAP) 20% above baseline during cross-clamping optimizes collateral cerebral blood flow and may reduce postoperative cognitive dysfunction.[113] Treatment is dependent upon the physician preference and includes the following options:[41, 52]

- Hypotension (in suspected hypovolemia): cautious volume expansion using isotonic crystalloid or colloid per physician choice
- Hypotension (in normovolemia): IV phenylephrine
- Hypertension: IV labetalol, nitroglycerin

3.2.8 Assist as indicated if immediate reopening of the artery is required. If the anesthesia is reversed and the patient is awakened in the operating room with new focal central

neurologic deficits; emergent re-exploration of the vessel may be done.[41] The artery will be evaluated for pulse flow and examined for any correctable flow-limiting causes.

3.2.9 Secure if present, any vacuum wound drains and assist with application of physician-determined type of sterile dressing (including topical sprays) over the operative incision site. There is no clear evidence that supports one type of dressing over another to prevent surgical site infections (SSI). The choice of dressing should be based on cost and symptom management goals[114] (Class I).

3.2.10 Assist anesthesia with ECG-monitored transport to the Post-Anesthesia Care Unit or other designated unit according to institutional protocol.

4. Postoperative Care

After surgical recovery has occurred, the patient is transferred to a secondary nursing unit until discharge. Depending upon institutional protocol, this may be an intensive care unit, a specialized cardiovascular/neurovascular or a vascular stepdown unit. Patients are usually monitored overnight in these same units with hospital discharge generally occurring the day after the procedure.[115]

4.1 Assessments

4.1.1. Obtain vital signs (BP, apical pulse, respirations, SpO2), level of consciousness, pain and sedation level at the specified intervals during recovery and progress thereafter according to institutional protocol. If the patient is transferred to a Post Anesthesia Care Unit (PACU), then use of a PACU scoring system is required (Class IV).[116] Maintain the systolic BP greater than 90mmHg and less than 180mmHg to avoid post-procedure complications of hypotension, hematoma formation and cerebral hyperperfusion syndrome.[52,117] Respirations should include rate, depth, effort and symmetry. If an arterial line is present, maintain system according to institutional policy. Pain and sedation levels should be assessed using a standardized scoring system. Sedation scale options include the Richmond-Agitation Sedation, Motor Activity, Sedation-Agitation or the Ramsey.[118] Pain is typically self-reported using a numeric or Faces scale.[119] There is a lack of evidence regarding the frequency in obtaining vital signs (VS) in the immediate post-procedure period. A single randomized controlled trial of 189 patients compared an experimental protocol (VS every 1 hour for 2 hours then every 4 hours for 24 hours) to standard practice (VS every 1 hour for 4 hours then every 4 hours for 24 hours) for post-operative patient monitoring. There were no significant differences observed between the two groups at 4 or 24 hours.[120] The authors recommend that clinician judgment should be used in monitoring VS frequency. The American Society of PeriAnesthesia Nurses (ASPAN) advises that VS frequency should be determined by each individual facility and pain should be assessed frequently.[116, 119] The ASPAN website reports that expert opinion states VS should be taken every 5 to 15 minutes during the initial stabilization and more frequently if clinically indicated.[116]

4.1.2. Perform a neurological assessment upon unit arrival and at scheduled time intervals throughout the remaining hospital stay to monitor for the development of stroke.

There is a lack of evidence-based research specific to the timing of neurological assessments after a CEA. A frequency option can be modified from the guidelines published in the American Heart Association nursing and interdisciplinary care of the acute ischemic stroke patient.[117] In those stroke patients receiving thrombolytic therapy, the recommendation for neurological assessments includes q15 minutes for the first two hours then progress to q1h for 16 hours. Further progression can be according to institutional protocol (typically every 4 hours until discharge). Minimally include: orientation, the Glasgow Coma Scale, pupil reaction to light, level of consciousness as described above and motor response. Other available stroke scales include; the Hunt and Hess Scale, NIHSS (National Institute of Health Stroke Scale) and Canadian Neurological Scale.[121] The choice of stroke scale is dependent upon the institution. Compare results with the preoperative assessments. Notify the physician of any decrease in these post-operative findings. This comparison allows for exposure of surgery-related neurologic sequelae.

4.1.3. Perform a cranial nerve assessment upon unit arrival and at scheduled time intervals with the neurological assessment throughout the remaining hospital stay to monitor for cranial nerve injury. Notify the surgeon if any occur. The cranial nerves most frequently at risk include VII (facial), IX (glossopharyngeal), X (vagus), XI (accessory) and XII (hypoglossal). Refer to Appendix A for a description of the relevant cranial nerves and associated assessments.

Cranial nerve injury is the most common complication following CEA with an incidence of 4-9%.[37, 41] This is due to the anatomic location of the specific cranial nerves in relationship to the carotid bifurcation. Injury is typically transient resolving in several days to weeks.

4.1.4. Assess the neck incision site upon unit arrival and at scheduled time intervals with the neurological assessment throughout the remaining hospital stay to monitor for development of a neck hematoma. If a bulb drain is present, measure and record the drainage every shift. Notify the physician immediately if a neck hematoma occurs or there is an increase in volume or change in drainage color (e.g. from a dark red to a bright red).

4.1.5 Administer oxygen via nasal cannula at flow rates as ordered. The goal is to maintain a target oxygen saturation of at least 94% in acutely ill patients and 92% in those patients at risk for hypercapnic respiratory failure unless otherwise specified (Class IV).[122]

4.1.6. Obtain a 12-lead ECG at the specified time per institutional protocol. Notify the surgeon of any abnormalities.
The incidence of postoperative myocardial infarction after CEA is approximately 2.3%.[37] In many patients, carotid artery disease is considered a marker for diffuse arterial disease including the coronary arteries.

4.1.7. Maintain a patent IV site and continuous ECG monitoring as specified by the physician. The potential for cardiac arrhythmias, coronary ischemia and other hemodynamic complications may develop in the post-operative period. Notify the surgeon if any ECG changes occur.

In addition to myocardial infarction, the potential for bradyarrhythmias may occur Research has demonstrated new onset atrial fibrillation is associated with intraoperative hypotension in the CEA patient [123] and those patients with a history of chronic atrial fibrillation had a higher risk of stroke and death from CEA.[124]

4.2 Patient Positioning and Activity Level

4.2.1 Elevate the head of the bed to 30-degrees or at physician-specified angle during the hospital stay. This promotes venous drainage and reduces intracranial pressure.[117] If a cervical block was used the patient is not able to eat or drink until the block wears off. The patient should be assessed for a pharyngeal reflex or gag reflex by touching the roof of the mouth, the back of the tongue, the area around the tonsils or the back of the throat. If a gag reflex is established the patient can begin to drink and eat without risk of aspiration. Maintaining head elevation to 30-degrees is also necessary until the pharyngeal reflex returns.

4.2.2 Maintain the patient on bed rest if ordered for the time as specified. There is an absence of research studies specifying the precise bed rest time, however many institutions keep the patient in bed while vasoactive medications are required for hemodynamic stability. Once the medications are discontinued, the patient may get out of bed. Another clinical practice option is to maintain bed rest the day of surgery and then ambulate the next day.[125]

4.3 Incisional Site Care

4.3.1 Remove any dressings if present, at the time interval as specified. The timing of dressing removal (within 48 hours or after 48 hours) does not appear to have a detrimental effect on outcomes[126] (Class I).

4.3.2 Keep the incision site dry for the first 48 hours after surgery. Avoid use of topical antimicrobial agents[127] (Class IV).

4.3.3 Maintain vacuum on the bulb drain if present. Remove the drain when ordered following institutional protocol. The type of drain and how the drain is secured should be verified before drain removal.[128]

4.4 Nutrition and Elimination

Once the patient has recovered from anesthesia, clear liquids are started. The patient is assessed for any swallowing difficulties. Diet is advanced to the preoperative diet either the evening of surgery or the next day once any complaints of nausea and vomiting have subsided. Complaints of dysphagia may require a bedside swallow evaluation to assess for aspiration.[129] Avoid unnecessary urethral catheterizations by following hospital protocols

based on Centers for Disease Control and Prevention (CDC) indications for urinary catheter insertion and using multimodal interventions to reduce the need for catheter use.[130, 131]

4.5 Assessment for Post-Operative Complications

Notify the physician immediately if any of the following occur:

4.5.1 Cranial nerve injury. As discussed under 4.1.3 in postoperative assessments, there are several cranial nerves that are at risk for injury due to their close proximity to the carotid bifurcation.

4.5.2 Neck hematoma or bleeding. The risks of neck hematoma include tracheal deviation and airway compromise. Cervical bleeding risk factors include postoperative hypertension, incomplete heparin reversal, DAPT and use of low-molecular weight dextran.[43,132,133] In the event of emergent airway compromise, the availability of fiberoptic intubation devices, suture removal kits and decompression of the airway by opening the incision may assist in tracheal intubation and airway stabilization.[134]

4.5.3 Hypotension. The treatment is the same as described under 3.2.7.

4.5.4 Hypertension. The treatment, blood pressure parameters and pathophysiological mechanisms behind the development of hypertension are the same as delineated under 3.2.7.

4.5.5 Cerebral hyperperfusion syndrome. Defined as an increase in cerebral blood flow associated with impaired cerebral autoregulation, the reported incidence of hyperperfusion syndrome ranges from 0.4% to 7.7% after a CEA and a mortality rate of up to 100% in some research.[41] Hyperperfusion syndrome typically occurs within two (2) weeks of the surgery.[37, 41] A change in the level of consciousness, confusion, and headache are often first seen. The headache is typically severe and may be located in the temporal, frontal, or orbital ipsilateral side resembling a migraine with or without nausea.[135, 136] There can be transient focal deficits or seizures without radiographic evidence of infarction from cerebral edema, and the most catastrophic complication; intracerebral hemorrhage.[37, 136] Risk factors include age older than 75 years, history of stroke, stenosis greater than 90%, contralateral stenosis, or a long history of hypertension.[136] The pathogenesis is thought to be the linking of two mechanisms: first, a state of maximal vasodilation of the cerebral arterioles in response to reduced perfusion from the carotid artery narrowing and second, a postprocedure increase in BP that leads to an increase in the cerebral blood flow. The impaired myogenic and neurogenic response of the cerebral arterioles is unable to compensate for this increase in blood volume leading to the cerebrovascular sequelae described above.[37, 41, 136] Strict blood pressure control in the postoperative period is the most important priority to prevent this occurrence.[37, 41, 135, 136]

4.5.6 Stroke

The incidence of stroke following CEA is 2.3%.[56] The major causes include technical issues, embolization from manipulation of the carotid artery, or inadequate flushing at the completion of the procedure.

4.5.7 Infection

The incidence of postoperative infection after carotid endarterectomy is less than 1% Healing difficulty may be seen in patients with a previously irradiated neck.[41] A systematic review by Knight[137] reported that CEA patch angioplasty using synthetic material was a potential source. The review noted that approximately 0.25-0.5% of Dacron patches became infected, with staphlycoccus aureus and staphylococcus epidermidis as primary organisms.[137]

4.5.8 General anesthesia-related complications

If the procedure performed under general anesthesia, the initial primary focus is on airway and breathing assessments. Airway and oxygenation are supported as indicated with airway positioning, oxygen as indicated, monitoring respiratory rate, depth and effort along with the oxygen saturation, nebulizer treatments, medications and oropharyngeal suctioning. Additional complications are treated per institutional protocol. The following complications from general anesthesia may be observed: [138, 139]

a. Upper airway obstruction from loss of pharyngeal muscle tone, edema, laryngospasm, neuromuscular blockade or history of obstructive sleep apnea.
b. Hypoxemia from alveolar hypoventilation or pulmonary edema.
c. Hemodynamic instability (hypotension/hypertension)
d. Nausea and/or vomiting
e. Shivering
f. Delirium

4.6 Diabetic Patient Management

In diabetic patients, provide glucose monitoring through a point-of-care device at intervals as specified per institutional protocol. Supplemental short-acting insulin may be administered during the day of the procedure to maintain a target glucose level between 140 and 180 mg/dL in these patients[92] (Class II). Hyperglycemia may potentiate the release of pro-inflammatory mediators.[140]

4.7 Postoperative Medications

Administer the following medications as ordered:

4.7.1 Antiplatelet therapy post CEA reduces the risk of stroke[141](Class I). Antiplatelet therapy with aspirin is recommended indefinitely[56] (Class IV). Beyond the first month, clopidogrel or combination low-dose aspirin and extended release dipyridamole may be administered as long-term prophylaxis against ischemic cardiovascular events[56] (Class IV). An optional consideration is evaluation for clopidogrel resistance. It has been reported that 16% to 50% of patients treated with clopidogrel have a heightened platelet reactivity (HPR) exposing the patient to the risk of acute vessel occlusion.[142] Identifying poor antiplatelet responders through

laboratory testing may allow for early adjustment of the treatment plan. However currently there is no standardized nationally recommended platelet function test that assesses clopidogrel resistance.[143]

4.7.2 Administer HMG-CoA reductase inhibitors (statins) daily at the time interval specified by the provider; or in cases of patient intolerance, use other agents that also target reduction in cholesterol and low-density lipoproteins (LDL) and increase in high density lipoproteins (HDL). Many manufacturers recommend a statin medication be taken at night because most cholesterol is synthesized when dietary intake is the lowest at this time.[144] HDL reduces the accumulation of foam cells in the artery wall by acting as a scavenger molecule and decreases platelet aggregation among other functions. [145]High-intensity statin treatment should be started in all patients under the age of 75 years with clinical atherosclerotic cardiovascular disease (ASCVD) unless contraindications are present[146] (Class IV). In patients over the age of 75 years, discussion should consider drug-drug interactions and patient preferences when initiating moderate or high intensity statin therapy[146] (Class IV). High intensity is defined as an approximate 50% or more reduction in LDL-C, moderate intensity lowers LDL-C by approximately 30% to 50%.[146] In those patients with familial history of heterozygous hypercholesterolemia or clinical ASCVD at maximal tolerated statin therapy, subcutaneous biweekly injections of alirocumab may be considered.[147]

4.7.3 Administer oral mild analgesic agents, such as acetaminophen, if indicated, based on reported pain level and institutional protocol. Consider avoidance of opioids that may not only mask/interfere with the neurologic assessment but also may increase intracranial pressure and decrease cerebral perfusion pressure[148] (Class III).

4.7.4 Assess for postoperative nausea and vomiting (PONV) and medicate with ordered antiemetic as indicated. The use of wrist acupoint P6 stimulation is another adjunctive treatment effective in reducing PONV[149] (Class I). There is no evidence supporting aromatherapy as an adjunct in treating PONV.[150] Nausea and vomiting can cause a transient or sustained rise in blood pressure which can lead to cerebral hyperperfusion syndrome.

4.7.5 Restart any home medications the patient is on such as antihypertensive agents at the pre-operative prescribed dose. If the patient is on warfarin or another Vitamin K antagonist (VKA), resume as ordered approximately 12 to 24 hours (the evening of or the next morning) after the surgery when there is adequate hemostasis[151] (Class I). Delay restart of VKA if hemostasis is uncertain or there are other reasons to delay therapy. If needed, consider a bridging treatment with low-molecular weight heparin administration until the VKA may be restarted.[151] Resumption of other oral antithrombotic agents such as dabigatran or rivaroxiban is dependent upon institutional protocol and verification of established hemostasis. There is no research recommending a precise restarting time for either of these oral antithrombotic agents after surgery.

4.8 Diagnostic Studies

Completion imaging (such as carotid duplex ultrasound) while the patient is hospitalized after CEA is per physician preference.[33] Schedule as ordered, follow up noninvasive imaging of the carotid arteries after discharge. Testing should be performed at scheduled intervals. Scheduling options may include initial evaluation in 30 days, 6 months and annually afterwards to assess for vessel patency[56] (Class IV). Once vessel patency stability has been established, vessel evaluation at longer time intervals may be considered.[56]

4.9 Patient Education

Provide discharge patient education regarding the following as indicated by institutional protocol and physician preference. This includes:

4.9.1 Continue prescribed medications with an emphasis on compliance as a means to reduce disease progression and the risk of development of complications.

4.9.2 Activity and incisional care to include the following

4.9.2.1 May shower after discharge at the time specified by the physician. Avoid use of lotions or ointments on the incision. Avoid rubbing the incision. Inspect the incision daily for signs of redness or drainage. Inform the patient that numbness along the jaw is common postoperatively and subsides within 6-12 months. Avoid clothing that rubs against the incision until it heals.[152] There is currently no evidence supporting or refuting benefits or harms from early versus delayed bathing in development of surgical site infections[153] (Class I).

4.9.2.2 Avoid driving until the neck incision has healed and the head can be turned without discomfort.[152]

4.9.2.3 Limiting the amount of weight the patient may lift is dependent upon physician preference. Clinically, patients are often cautioned against lifting anything heavier than 10 pounds for 48 hours after discharge.[154]

4.9.3 Signs and symptoms to report to the physician:[152]

4.9.3.1 Reoccurrence of neurologic symptoms or new neurological signs such as dizziness, paresthesias, vision changes or dysphagia. Several potential reasons for these may include disease progression, emboli/thrombus or technical issues such as vessel kinking.

4.9.3.2 Worsening headache that is similar to a migraine, which could be a sign of cerebral hyperperfusion syndrome (as described in Hyperperfusion syndrome). Early recognition of reperfusion syndrome can prevent significant negative outcomes.

4.9.3.3 Cardiopulmonary: dyspnea, chest pain, peripheral edema, productive sputa

4.9.3.4 Signs of infection: fever, incisional changes

4.9.4 Management of modifiable risk factors:

4.9.4.1 Smoking cessation counseling/adjunctive options. It has been demonstrated that patients who are current smokers have greater internal and common carotid wall thickening and internal carotid stenosis compared to non-smokers[155,156] (Class III).

4.9.4.2 Glycemic control in patients with diabetes as measured by the glycosylated hemoglobin level (A1c) to be maintained below or around 7%. (Class IV).[92] A high A1c level was found to be an independent predictor of intimal hyperplasia development leading to restenosis.[157]

4.9.4.3 Hypertension management to reduce the risk of postoperative cerebral hemorrhage and hyperperfusion syndrome as described above and to reduce disease progression. Hypertension increases intimal and medial arterial thickening, thus reducing blood flow[158] (Class II). In the general population, for patients 60 years or older, initiate pharmacologic treatment to lower the BP to a treatment goal of SBP <150mmHg and DBP <90mmHg[159] (Class IV). Provide education on the following lifestyle modifications: smoking cessation, weight reduction, limit sodium intake, avoid use of illicit drugs (cocaine, methamphetamines), limit alcohol consumption, increase physical activity level and stress reduction[160] (Class IV).

4.9.4.4 Cholesterol reduction through dietary modifications, disease control and medication compliance to reduce the risk of atherosclerotic disease progression and restenosis.[146]

4.10 Outcomes Reporting

Assist in data collection and reporting of outcomes as indicated based on the institutional quality improvement requirements. Utilize a standardized registry such as the Vascular Quality Initiative from the Society for Vascular Surgery or the Cardiovascular Data Registry Carotid Artery Revascularization and Endarterectomy Registry from the American Heart Association.[161] This will allow for assessment of risk-adjusted outcomes and benchmarking of institutional metrics against national results.

References

1. Treat-Jacobson DJ, Rich K, DeVeaux T, et al. Society for vascular nursing clinical practice guideline (CPG) for carotid artery stenting. *Journal of Vascular Nursing*. 2013;31(1):32-55.

2. Melnyk B. FE. Making the case for evidence-based practice and cultivating a spirit of inquiry. In: *Evidence-based nursing practice in nursing and healthcare: A guide to best practice.* 1st ed. Philadelphia: Lippincott, Williams & Wilkins; 2005:3-24.

3. American Association of Neuroscience Nurses, ed. *Care of the patient with aneurysmal subarachnoid hemorrhage.* ; 2007.

4. Fugate, M. & Perler, B. Contemporary carotid endarterectomy results in the united states. in: Modern trends in vascular surgery carotid artery disease. . . 2010:199-208.

5. DeBakey ME. Successful carotid endarterectomy for cerebrovascular insufficiency. nineteen-year follow-up. *JAMA (Chicago, Ill.)*. 1975;233(10):1083-1085. doi: 10.1001/jama.1975.03260100053020.

6. Benjamin E, Blaha M, Chiuve S. et al. Heart disease and stroke statistics--2017 update: A report from the american heart association. *Circulation*. 2017;135: e146-e603. doi: org/10.1161/CIR.0000000000000485.

7. Patel M, Greiner M, DeMartino L, et al. Geographic variation in carotid revascularization among medicare beneficiaries, 2003-2006. *Arch Intern Med*. 2010;170(14):1218-1225.

8. Ovbiagele B, Goldstein LB, Higashida RT, Howard VJ, et.al. Forecasting the future of stroke in the united states: A policy statement from the american heart association and american stroke association. *Stroke*. 2013;44(8):2361-2375.

9. White CJ, Beckman JA, Cambria RP, et al. Atherosclerotic peripheral vascular disease symposium II: Controversies in carotid artery revascularization. *Circulation*. 2008;118(25):2852-2959.

10. Agur A. Grant's atlas of anatomy. In: Agur, A., Lee, M., Agur, A.M., ed. *Grant's atlas of anatomy.* 12th ed. Philadelphia: Lippincott Williams & Wilkins; 2009:745-810.

11. Waxman S. Vascular supply of the brain. In: Waxman S, ed. *Clinical neuroanatomy.* ; 2010.

12. Mohrman DE. *Cardiovascular physiology.* New York: New York : Lange Medical Books/McGraw-Hill; 2010.

13. Hall J. Nervous regulation of the circulation and rapid control of arterial pressure. In: *Guyton and hall textbook of medical physiology.* 12th ed. Philadelphia: Saunders; 2011.

14. Rapp JH, Owens C, Johnson M. Blood vessels and lymphatic disorders. In: McPhee S.J. PMA, ed. *Current medical diagnosis and treatment.* ; 2011.

15. Bates ER, Babb JD, Casey DE,Jr, et al. ACCF/SCAI/SVMB/SIR/ASITN 2007 clinical expert consensus document on carotid stenting. *Vascular Medicine*. 2007;12(1):35-83. http://ovidsp.ovid.com/ovidweb.cgi?T=JS&CSC=Y&NEWS=N&PAGE=fulltext&D=med4&AN=17451093;

16. Penn M. CG. Lipoprotein oxidation, arterial inflammation, and atherogenesis. In: Fuster V, Topol E., Nabel E., ed. *Atherothrombosis and coronary artery disease.* 2nd ed. Philadelphia: Lippincott, Williams & Wilkins; 2005:111.

17. Lopez-Yunez A. Obstructive carotid artery disease and evidence-based benefits of revascularization. In: Al-mubarak N, Roubin G, Iyer S, Vitek J, eds. *Carotid artery stenting: Current practice and techniques.* 1st ed. Philadelphia: Lippincott Williams & Wilkins; 2004.

18. Redgrave JNE, Lovett JK, Gallagher PJ, Rothwell PM. Histological assessment of 526 symptomatic carotid plaques in relation to the nature and timing of ischemic symptoms: The oxford plaque study. *Circulation*. 2006;113(19):2320-8. doi: 10.1161/CIRCULATIONAHA.105.589044.

19. White C, Jaff M. Extracranial carotid artery disease. In: White C, Jaff M, eds. *Vascualr disease: Diagnostic and therapeutic approaches.* 1st ed. Minneapolis: Cardiotext Publlishing; 2011:73-98.

20. Kapadia S. YJ. Nonsurgical management of carotid artery disease. In: Fuster V, Topol E., Nabel E., ed. *Atherothrombosis and coronary artery disease.* 2nd ed. Philadelphia: Lippincott, Williams & Wilkins; 2005:1551-1562.

21. North American Symptomatic Carotid Endarterectomy Trial (NASCET) Steering Committee. North American symptomatic carotid endarterectomy trial: Methods, patient characteristics, and progress. *Stroke*. 1991;22:711-720.

22. Jensen FE, Creager MA. Cerebrovascular disease. In: Creager MA, ed. *Vascular disease.* ; 2003.

23. Ferguson G, Eliasziw M, Barr H, et.al. The North American symptomatic carotid endarterectomy trial surgical results in 1415 patients. *Stroke*. 1999;30:1751-1758.

24. European Carotid Surgery Trialists' Collaborative Group. Randomised trial of endarterectomy for recently symptomatic carotid stenosis: Final results of the MRC European carotid surgery trial (ECST). *Lancet*. 1998;351:1379-1387.

25. Rothwell P, Gutnikov S, Warlow C. Reanalysis of the final results of the randomized carotid surgery trial. . *Stroke*. 2003;34:514-523.

26. Hobson RW2, Weiss DG, Fields WS, et al. Efficacy of carotid endarterectomy for asymptomatic carotid stenosis. the veterans affairs cooperative study group. *N Engl J Med*. 1993;328(4):221-227.

27. Executive committee for the Asymptomatic Carotid Atherosclerosis Study (ACAS). Endarterectomy for asymptomatic carotic artery stenosis. *JAMA*. 1995;273:1421-1502.

28. White CJ, Beckman JA, Cambria RP, et al. Atherosclerotic peripheral vascular disease symposium II: Controversies in carotid artery revascularization. *Circulation*. 2008;118(25):2852-2859..

29. Halliday A, Mansfield A, Marro J, et al. Prevention of disabling and fatal strokes by successful carotid endarterectomy in patients without recent neurological symptoms: Randomised controlled trial. *Lancet*. 2004;363(9420):1491-1502. doi: 10.1016/S0140-6736(04)16146-1.

30. Chambers BR, Donnan G. Carotid endarterectomy for asymptomatic carotid stenosis. *Cochrane Database of Systematic Reviews*. 2005(4).

31. Beckman JA. Management of asymptomatic internal carotid artery stenosis. *JAMA*. 2013;310(15):1612-1618.

32. O'Brien M, Chandra A. Carotid revascularization: Risks and benefits. *Vascular Health & Risk Management*. 2014;10:403-416.

33. Ricotta JJ, Aburahma A, Ascher E, et al. Updated Society for Vascular Surgery guidelines for management of extracranial carotid disease: Executive summary. *Journal of Vascular Surgery*. 2011;54(3):832-836.

34. Paraskevas KI, Mikhailidis DP, Veith FJ, Spence JD. Definition of best medical treatment in asymptomatic and symptomatic carotid artery stenosis. *Angiology*. 2016;67(5):411-419.

35. Moore WS. Issues to be addressed and hopefully resolved in the carotid revascularization endarterectomy versus stenting trial 2. *Angiology*. 2016;67(5):408-410..

36. Bevilacqua S, Romagnoli S, Ciappi F, et.al. Anesthesia for carotid endarterectomy: The third option. patient cooperation during general anesthesia. *Anesth Analg*. 2009;108:1929-1936.

37. Beaulieu R, Abularrage C. Carotid endarterectomy. *Current Surgical Therapy, 11th Edition*. 2014:811-817.

38. Rerkasem K, Rothwell PM. Routine or selective carotid artery shunting for carotid endarterectomy (and different methods of monitoring in selective shunting). *Cochrane Database of Systematic Reviews*. 2009(4).

39. Cao P, De Rango P, Zannetti S, Giordano G, Ricci S, Celani MG. Eversion versus conventional carotid endarterectomy for preventing stroke. *Cochrane Database of Systematic Reviews*. 2000(4).

40. Rerkasem K, Rothwell PM. Patches of different types for carotid patch angioplasty. . *Cochrane Database of Systematic Reviews*. 2010(3).

41. Roseborough G, Perler B. Carotid artery: Endarterectomy. . *Rutherford's Vascular Surgery, 8th Edition*. 2014:1514-1543.

42. Dellagrammaticas D, Lewis S, Gough M. Is heparin reversal with protamin after carotid endarterectomy dangerous? *Eur J Vasc Endovasc Surg*. 2008;36(1):41-44.

43. Stone D, Nolan B, Schanzer A, et.al. Protamine reduces bleeding complications associated with carotid endarterectomy without increasing risk of stroke. *J Vasc Surg*. 2010;51(3):559-564.

44. Farber A, Tan T, Rybin D, et al. Intraoperative use of dextran is associated with cardiac complications after carotid endarterectomy. *Journal of Vascular Surgery*. 2013;57(3):635-641.

45. Probasco J, Sahin B, Tran T, et.al. The preoperative neurological evaluation. . *Neurohospitalist*. 2013;3(4):209-220.

46. American Academy of Allergy Asthma and Immunology. Position statement: The risk of severe allergic reactions from the use of potassium iodide for radiation emergencies. https://www.aaaai.org/Aaaai/media/MediaLibrary/PDF%20Documents/Practice%20and%20Parameters/Potassium-iodide-in-radiation-emergencies-2004.pdf. Updated 2004. Accessed 03/18/2017.

47. Christiansen C. X-ray contrast media--an overview. *Toxicology*. 2005;209(2):185-187.

48. Beaty AD, Lieberman PL, Slavin RG. Seafood allergy and radiocontrast media: Are physicians propagating a myth?. *Am J Med*. 2008;121(2):158.e1-158.e4.

49. Pickering TG, Hall JE, Appel LJ, et al. Recommendations for blood pressure measurement in humans and experimental animals: Part 1: Blood pressure measurement in humans: A statement for professionals from the subcommittee of professional and public education of the american heart association council on high blood pressure research. *Circulation*. 2005;111(5):697-716.

50. Spittel P. Subclavian steal syndrome. http://www.uptodate.com/contents/subclavian-steal-syndrome. Updated 2016. Accessed 03/17/2017.

51. Eagleton MJ, Kang J. Preoperative management. . *Rutherford's Vascular Surgery 8th Edition*. 2014:466-479.

52. Varon J, Marik P. Perioperative hypertension management. . *Vasc Health Risk Manag*. 2008;4(3):615-627.

53. Seidel H, Ball J, Dains J, et.al. Objective data - physical findings. In: Seidel, H., Ball, J., Dainns, J., Flynn, J., Solomon, B., Strward R, eds. *Physical examination handbook.* 7th Ed. ed. St. Louis: Mosby-Elsevier; 2010:306-314.

54. Sivanaser V, Manninen P. Preoperative assessment of adult patients for intracranial surgery. . *Anesthesiol Res Pract*. 2010.

55. NIH stroke scale international. http://www.nihstrokescale.org/. Updated 4/11/2012. Accessed 3/20/17.

56. Brott TG, Halperin JL, Abbara S, et al. 2011 ASA/ACCF/AHA/AANN/AANS/ACR/ASNR/CNS/SAIP/SCAI/SIR/SNIS/SVM/SVS guideline on the management of patients with extracranial carotid and vertebral artery disease: Executive summary. *Circulation*. 2011;124(4):489-532.

57. Busuttil SJ, Franklin DP, Youkey JR, Elmore JR. Carotid duplex overestimation of stenosis due to severe contralateral disease. *Am J Surg*. 1996;172(2):144-147.

58. Comerota AJ, Salles-Cunha SX, Daoud Y, Jones L, Beebe HG. Gender differences in blood velocities across carotid stenoses. *Journal of Vascular Surgery*. 2004;40(5):939-944.

59. Lal BK, Hobson RW,2nd, Tofighi B, Kapadia I, Cuadra S, Jamil Z. Duplex ultrasound velocity criteria for the stented carotid artery. *Journal of Vascular Surgery*. 2008;47(1):63-73.

60. Nederkoorn PJ, van der Graaf Y, Hunink MG. Duplex ultrasound and magnetic resonance angiography compared with digital subtraction angiography in carotid artery stenosis: A systematic review. *Stroke*. 2003;34(5):1324-1332.

61. Yuan C, Mitsumori LM, Ferguson MS, et al. In vivo accuracy of multispectral magnetic resonance imaging for identifying lipid-rich necrotic cores and intraplaque hemorrhage in advanced human carotid plaques. *Circulation*. 2001;104(17):2051-2056.

62. Remonda L, Senn P, Barth A, Arnold M, Lovblad K, Schroth G. Contrast-enhanced 3D MR angiography of the carotid artery: Comparison with conventional digital subtraction angiography. *Ajnr: American Journal of Neuroradiology*. 2002;23(2):213-219.

63. Gronholdt ML. B-mode ultrasound and spiral CT for the assessment of carotid atherosclerosis. *Neuroimaging Clin N Am*. 2002;12(3):421-435.

64. Wardlaw JM, Chappell FM, Best JJ, Wartolowska K, Berry E, NHS Research and Development Health Technology Assessment Carotid Stenosis Imaging,Group. Non-invasive imaging compared with intra-arterial angiography in the diagnosis of symptomatic carotid stenosis: A meta-analysis. *Lancet*. 2006;367(9521):1503-1512.

65. Willinsky R, Taylor S, terBrugge K, et.al. Neurologic complications of cerebral angiography: Prospective analysis of 2,899 procedures and review of the literature. . *Neuroradiology*. 2003;227(2):522-528.

66. Iyer S.S., Al-Mubarak N, Vitek JJR, G. S. Procedural techniques. In: Al-Mubarak N, Ruobin GS, Iyer S, eds. *Carotid artery stenting: Current practice and tequniques.* 1st ed. Philadelphia: Lippincott Williams & Wilkins; 2004:105-123.

67. Fleisher LA. American College of Cardiology/American Heart Association. Cardiac risk stratification for noncardiac surgery: Update from the American College of Cardiology/American Heart Association 2007 guidelines. *Cleve Clin J Med*. 2009;76(Suppl 4):S9-15.

68. Fleischmann KE, Beckman JA, Buller CE, et al. 2009 ACCF/AHA focused update on perioperative beta blockade: A report of the American College of Cardiology Foundation/American Heart Association task force on practice guidelines. *Circulation*. 2009;120(21):2123-2151.

69. Poirer P, Alpert M, Fleisher L, et.al. Cardiovascular evaluation and management of severely obese patients undergoing surgery. . *Circulation*. 2009;120:86-95.

70. American Society of Anesthesiologists Task Force. Practical advisory for preanesthesia evaluation. *Anesthesiology*. 2012;116(3):1-17.

71. Raju I, Fraser K. Anesthesia for carotid surgery.
. *Anesth Intens Care*. 2013;14(5):208-211.

72. Joanna Briggs Institute. Preoperative hair removal to reduce surgical site infection. *Best Practice*. 2007;11(4):1-4.

73. Tanner J, Woodings D, Moncaster K. Preoperative hair removal to reduce surgical site infection. *Cochrane Database of Systematic Reviews*. 2006;3:004122.

74. Edminston C, Okoli O, Graham M, et.al. Evidence for using chlorhexidine gluconate preoperative cleansing to reduce the risk of surgical site infection. *AORN*. 2010;92(5):509-518.

75. American Society of Anesthesiologists Committee on Standards and Practice Parameters. Practice guidelines for preoperative fasting and the use of pharmacologic agents to reduce the risk of pulmonary aspiration: Application to healthy patients undergoing elective procedures. . *Anesthesiology*. 2011;114(3):495-511.

76. Blackburn K, Neaton M. Redesigning the care of carotid endarterectomy patients . *J Vasc Nurs*. 1997;15(1):8-12.

77. Kruzik N. Benefits of preoperative education for adult elective surgery patients. . *AORN*. 2009;90(3):381-387.

78. Whinney C. Perioperative medication management: General principles and practical applications. *Clev Clinic J Med*. 2009;76(4):S126-S132.

79. Kuwajerwala N, Schwer W. Perioperative medication management. . http://emedicine.medscape.com/article/284801-overview Web site. Updated 2015. Accessed 3/17/2017

80. Stoner M, deFrietas D. Process of care for carotid endarterectomy: Perioperative medical management. *J Vasc Surg*. 2010;52(1):223-231.

81. Dimitrova G, Tulman D, Bergese S. Perioperative management of antiplatelet therapy in patients with drug-eluting stents. *HSR Proc Intensive Care Cardiovasc Anesth*. 2012;4:153-167.

82. Capodanno D, Angiolillo D. Management of antiplatelet therapy in patients with coronary artery disease requiring cardiac and noncardiac surgery . *Circulation*. 2013;128:2785-2798.

83. Fleming M, Stone W, Scott P, et.al. Safety of carotid endarterectomy in patients concurrently on clopidogrel. . *Ann Vasc Surg*. 2009;23(5):612-615.

84. Wait S, Abla A, Killory B., et.al. Safety of carotid endarterectomy while on clopidogrel. . *J Neurosurgery*. 2010;113:908-912.

85. Perler BA. The effect of statin medications on perioperative and long-term outcomes following carotid endarterectomy or stenting. *Semin Vasc Surg*. 2007;20(4):252-258.

86. De Martino R, Elrup-Jorgensen J, Nolan B, et.al. Preoperative management with antiplatelet and statin medication is associated with reduced mortality following vascular surgery. . *J Vasc Surg*. 2014;59(6):1615-1621.

87. National Cholesterol Education Program Expert Panel. Detection, evaluation and treatment of high blood cholesterol in adults final report. *Circulation*. 2002;106(25):3143-3421.

88. Howell S. Carotid endarterectomy. *Br J Anesth*. 2007;99:119-131.

89. Stoneham MD, Thompson JP. Arterial pressure management and carotid endarterectomy. . *Br J Anesth*. 2009;102(4):442-452.

90. Fleisher LA, Fleischmann KE, Auerbach AD, et al. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: A report of the American College of Cardiology/American Heart Association task force on practice guidelines. *Circulation*. 2014;130(24):e278-333.

91. Donner T, Flammer K. Diabetes management in the hospital. *Med Clin N Am*. 2008;92:407-425.

92. American Diabetes Association. Standards of medical care in diabetes--2014. *Diabetes Care*. 2014;37(Suppl 1):S14-80.

93. Muluk V, MacPherson D, Cohn S, Whinney C. Perioperative medication management. . http://www.uptodate.com/contents/perioperative-medication-management?source=search_result&search=nonsteroidal+antiinflammatory+drugs+and+surgery&selectedTitle=1~150#H28. Updated 2017; accessed 3/17/2017.

94. Saber W. Perioperative medication management: A case-based review of general principles. *Clev Clin J Med*. 2006;73(Suppl1):S82-S87.

95. Douketis J, Spyropoulos A, Spencer F, et al. Perioperative management of antithrombotic therapy: Antithrombotic therapy and prevention of thrombosis, 9th ed: American college of chest physicians evidence-based clinical practice guidelines. *Chest*. 2012;141(2 Suppl):e326S-e350S. doi: 10.1378/chest.11-2298.

96. *Pradaxa (dabigatran) product monograph.* Boehringer Ingelheim Pharmaceuticals, Inc.; 2012.

97. *Xarelto (rivaroxaban) product monograph.* Janssen Pharmaceuticals, Inc.; 2011.

98. *Eliquis (apixiban) product monograph.* Bristol-Meyers Squibb; 2012.

99. *Savaysa (edoxaban) product monograph.* Daiichi Sankyo Co., LTD; 2015.

100. Skalli S, Zaid A, Soulaymani R. Drug interactions with herbal medicines. *Ther Drug Monit*. 2007;29(6):679-686.

101. Schurks M, Glynn RJ, Rist PM, Tzourio C, Kurth T. Effects of vitamin E on stroke subtypes: Meta-analysis of randomised controlled trials. *BMJ*. 2010;341:5702.

102. Gardiner P, Phillips R, Shaughnessy AF. Herbal and dietary supplement--drug interactions in patients with chronic illnesses. *Am Fam Physician*. 2008;77(1):73-78.

103. Yue QY, Bergquist C, Gerden B. Safety of st john's wort (hypericum perforatum). *Lancet*. 2000;355(9203):576-577.

104. World Health Organization. WHO guidelines for safe surgery. http://www.who.int/patientsafety/safesurgery/tools_resources/9789241598552/en/ . Web site. Accessed 3/17/2017.

105. Lyons V, Popejoy L. Meta-analysis of surgical safety checklist effects on teamwork, communication, morbidity, mortality and safety. . *Western J Nurs Res*. 2014;36:245-261.

106. Joint Commission. National patient safety goals. https://www.jointcommission.org/assets/1/6/2017_NPSG_HAP_ER.pdf. Updated 2017. Accessed 3/21/17.

107. Arnold M, Perler B. Carotid artery: Endarterectomy - chapter 100. In: *Rutherfords Vascular Surgery 8th edition.* Vol 2. Elsevier; 2014:1514-1543.

108. Bratzler D, Dellinger E, Olsen K. ASHP report: Clinical practice guidelines for antimicrobial prophylaxis in surgery. *Am J Health-Syst Pharm*. 2013;70:195-283.

109. Malignant Hyperthermia Association of the United States. Anesthetic agent choice for the mh-susceptible patient. http://medical.mhaus.org/index.cfm/fuseaction/Content.Display/PagePK/AnestheticList.cfm. Updated 2017. Accessed 3/17/2017.

110. Glahn KP, Ellis FR, Halsall PJ, et al. Recognizing and managing a malignant hyperthermia crisis: Guidelines from the European malignant hyperthermia group. *Br J Anaesth*. 2010;105(4):417-420.

111. Pennekamp CW, Moll FL, & de Borst GJ. The potential benefits and the role of cerebral monitoring in carotid endarterectomy. *Curr Opin Anaesthesiol*. 2011;24(6):693-697.

112. Ali A, Green D, Zayed H, et al. Cerebral monitoring in patients undergoing carotid endarterectomy using a triple assessment technique. *Interact Cardiovasc Thorac Surg*. 2011;12:454-457.

113. Heyer E, Mergeche J, Anastasian Z, et al. Arterial blood pressure management during carotid endarterectomy and early cognitive dysfunction. *Neurosurg*. 2014;74(3):245-253.

114. Dumville JC, Gray TA, Walter CJ, Sharp CA, Page T. Dressings for the prevention of surgical site infection. *Cochrane Database of Systematic Reviews*. 2014(9).

115. Glaser J, Kuwayama D, Stone D, et al. Factors that determine the length of stay after carotid endarterectomy represent opportunities to avoid financial losses. *Journal of Vascular Surgery*. 2014;60(4):966-72.e1.

116. American Society of PeriAnesthesia Nurses. *Perianesthesia nursing standards and practice recommendations 2010-2012.* Chery Hill, NJ: ASPAN; 2010.

117. Summers D, Leonard A, Wentworth D, et al. Comprehensive overview of nursing and interdisciplinary care of the acute ischemic stroke patient. A scientific statement from the american heart association. *Stroke*. 2009;40:2911-2944.

118. Abbott/American Association of Critical-Care Nurses. Saint Thomas Health System Sedation Expert Panel Members. Consensus conference on sedation assessment. A collaborative venture by abbott laboratories, american association of critical-care nurses, and saint thomas health system. *Crit Care Nurse*. 2004;24(2):33-41.

119. Krenzischek DA, Wilson L, Aspan. An introduction to the ASPAN pain and comfort clinical guideline. *Journal of PeriAnesthesia Nursing*. 2003;18(4):228-236.

120. Fernandez R, Griffiths R. A comparison of an evidence based regime with the standard protocol for monitoring postoperative observation: A randomised controlled trial. *Australian Journal of Advanced Nursing*. 2005;23(1):15-21.

121. Internet Stroke Center & National Institute of Health. Stroke assessment scales. http://www.strokecenter.org/professionals/stroke-diagnosis/stroke-assessment-scales/. Updated 2015. Accessed 3/17/2017.

122. O'Driscoll B, Howard L, Davison A. BTS guideline for emergency oxygen use in adult patients. *Thorax*. 2008;68(Suppl VI):vi1-vi68.

123. Sposato L, Suarex A, Jaurequi A, et al. Intraoperative hypotension, new onset atrial fibrillation and adverse outcome after carotid endarterectomy. *J Neurol Sci*. 2011;309(1-2):5-8.

124. Harthun N, Stukenborg G. Atrial fibrillation is associated with increased risk of perioperative stroke and death from carotid endarterectomy. *J Vasc Surg*. 2010;51(2):330-336.

125. Chung J, Dodson T. Carotid endarterectomy. In: Lubin M, Dodson T, Winawer N, eds. *Medical management of the surgical patient.* 5th ed. United Kingdom: Cambridge University Press; 2013:605-607.

126. Toon CD, Ramamoorthy R, Davidson BR, Gurusamy KS. Early versus delayed dressing removal after primary closure of clean and clean-contaminated surgical wounds. *Cochrane Database of Systematic Reviews*. 2013(9).

127. Orsted H, Keast D, Kuhnke J, et al. Best practice recommendations for the prevention and management of open surgical wounds. *Wound Care Canada*. 2010;8(1):6-34.

128. Makic M. Drain removal. In: Wiegand D, ed. *AACN procedure manual for critical care.* 6th ed. Philadelphia: Elsevier; 2011:1172-1174.

129. Masiero S, Previato C, Addante S, et al. Dysphagia in post-carotid endarterectomy: A prospective study. *Ann Vasc Surg*. 2007;21(3):318-320.

130. Centers for Disease Control and Prevention. Guideline for prevention of catheter-associated urinary tract infections. http://www.cdc.gov/hicpac/cauti/02_cauti2009_abbrev.html. Updated 2009. Accessed 3/20/17.

131. Meddings J, Krein S, Fakih M, et al. Chapter 9: Reducing unnecessary urinary catheter use and other strategies to prevent catheter-associated urinary tract infections: Brief update review. Making Healthcare Safer II: An updated critical analysis of the evidence for patient safety practices. Evidence Reports/Technology Assessments No 211. Web site. http://www.ncbi.nlm.nih.gov/books/NBK133354/#ch9.s3. Updated 2013. Accessed 3/17/2017.

132. Comerota A, DiFiore R, Tzilinis A, Chahwan S. Cervical hematoma following carotid endarterectomy is morbid and preventable - a 12 year case-controlled review. *Vasc Endovasc Surg*. 2012;46(8):610-616.

133. Gisbert S, Almonacil V, Garcia J, et al. Predictors of cervical bleeding after carotid endarterectomy. *Ann Vasc Surg*. 2014;28(2):366-374.

134. Shakespeare WA, Lanier WL, Perkins WJ, Pasternak JJ. Airway management in patients who develop neck hematomas after carotid endarterectomy. *Anesth Analg*. 2010;110(2):588-593.

135. Moulakakis KG, Mylonas SN, Sfyroeras GS, Andrikopoulos V. Hyperperfusion syndrome after carotid revascularization. *Journal of Vascular Surgery*. 2009;49(4):1060-1068.

136. Adhiyaman V, Alexander S. Cerebral hyperperfusion syndrome following carotid endarterectomy. *Q J Med*. 2007;100:239-244.

137. Knight BC, Tait WF. Dacron patch infection following carotid endarterectomy: A systematic review of the literature. *Eur J Vasc Endovasc Surg*. 2009;37(2):140-148.

138. Barone C, Pablo c, Baron G. Postanesthetic care in the critical care unit. *Crit Care Nurse*. 2004;24(1):38-45.

139. Nicholau D. The postanesthesia care unit. In: Miller R, Erickson L, Fleisher L, Wiener-Kronish J, Young W, eds. *Miller's anesthesia.* 7th ed. Orlando: Churchill Livingstone; 2009:2707-2728.

140. Dandona P, Aljada A, Chaudhuri A, Bandyopadhyay A. The potential influence of inflammation and insulin resistance on the pathogenesis and treatment of atherosclerosis-related complications in type 2 diabetes. *Journal of Clinical Endocrinology & Metabolism*. 2003;88(6):2422-2429.

141. Engelter S, Lyrer P. Antiplatelet therapy for preventing stroke and other vascular events after carotid endarterectomy. *J Clin Endocrinol Meta*. 2003;88(6):2422-2429.

142. Tantry U, Hennekens C, Zehnder J. Clopidogrel resistance an clopidogrel treatment failure. UpToDate Web site. http://www.uptodate.com/contents/clopidogrel-resistance-and-clopidogrel-treatment-failure#H2. Updated 2016. Accessed 3/17/2017.

143. Nguyen TA, Diodati JG, Pharand C. Resistance to clopidogrel: A review of the evidence. *J Am Coll Cardiol*. 2005;45(8):1157-1164.

144. Wallace A, Chinn D, Rubin G. Taking simvastatin in the morning compared with in the evening: Randomised controlled trial. *BMJ*. 2003;327(7418):788.

145. Barter PJ, Nicholls S, Rye KA, Anantharamaiah GM, Navab M, Fogelman AM. Antiinflammatory properties of HDL. *Circ Res*. 2004;95(8):764-772.

146. Stone N, Robinson J, Lichtenstein A, et al. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults. *J Am Coll Cardiol*. 2014;63(25):2889-2934.

147. *Praluent product monograph.* Regeneron Pharmaceuticals, Inc.; 2015.

148. de Nadal M, Munar F, Poca MA, Sahuquillo J, Garnacho A, Rossello J. Cerebral hemodynamic effects of morphine and fentanyl in patients with severe head injury: Absence of correlation to cerebral autoregulation. *Anesthesiology*. 2000;92(1):11-19.

149. Lee A, Fan LTY. Stimulation of the wrist acupuncture point p6 for preventing postoperative nausea and vomiting. *Cochrane Database of Systematic Reviews*. 2009(2).

150. Hines S, Steels E, Chang A, Gibbons K. Aromatherapy for treatment of postoperative nausea and vomiting. *Cochrane Database of Systematic Reviews*. 2012(4).

151. Douketis J.D., Spyropolous, A., Spencer, F., Mayr M, et al. Perioperative management of antithrombotic therapy. *Chest*. 2012(141):e326-e350S.

152. U.S. National Library of Medicine - National Institute of Health. Carotid artery surgery - discharge. http://www.nlm.nih.gov/medlineplus/ency/patientinstructions/000238.htm. Updated 2015. Accessed 3/17/2017.

153. Toon CD, Sinha S, Davidson BR, Gurusamy KS. Early versus delayed post-operative bathing or showering to prevent wound complication. *Cochrane Database of Systematic Reviews*. 2013(10).

154. Hamel WJ. Femoral artery closure after cardiac catheterization. *Crit Care Nurse*. 2009;29(1):39-46.

155. Tell GS, Polak JF, Ward BJ, Kittner SJ, Savage PJ, Robbins J. Relation of smoking with carotid artery wall thickness and stenosis in older adults. the cardiovascular health study. the cardiovascular health study (CHS) collaborative research group. *Circulation*. 1994;90(6):2905-2908.

156. Liang Y, Shiel LM, Teede H, Kotsopoulos, D., McNeil, J., Cameron JD, McGrath BP. Effects of bloood pressure, smoking, and their interaction on carotid artery structure and function. *Hypertension*. 2001;37:6-11.

157. Willfort-Ehringer A, Ahmadi R, Gessl A, et al. Neointimal proliferation within carotid stents is more pronounced in diabetic patients with initial poor glycaemic state. *Diabetologia*. 2004;47(3):400-406.

158. Dobrin PB. Mechanical factors associated with the development of intimal and medial thickening in vein grafts subjected to arterial pressure. A model of arteries exposed to hypertension. *Hypertension*. 1995;26(1):38-43.

159. James P, Oparil S, Carter B, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: Report for the panel members appointed to the eigth joint national committee. *JAMA*. 2014;311(5):507-520.

160. Weber M, Schiffrin E, White W, et al. Clinical practice guidelines for the management of hypertension int he community. *J Clin Hypertens*. 2014;16(1):14-26.

161. Wimmer N, Spertus J, Kennedy K, et al. Clinical prediction model suitable for assessing hospital quality for patients undergoing carotid endarterectomy. *J Am Heart Assoc*. 2014.

Appendix A: Nursing Activities Checklist in Caring for CEA Patient

CEA Nursing Activities**			
Pre-Operative	**Yes**	**No**	**NA**
• Assess for co-morbid conditions, risk factors & previous carotid/neck surgery. Notify MD if present			
• Obtain baseline vital signs (BP both arms, apical/radial pulse, respirations, SpO_2, temperature)			
• Obtain list of current medications & assess for any allergies			
• Perform a targeted physical assessment. The neurological component to include speech, pupils, facial symmetry, gait, Glasgow coma scale, NIHSS, cranial nerves. Notify MD of abnormal results.			
• Verify ordered diagnostic tests are done. Notify MD of any abnormal results.			
• Start 1-2 peripheral IV sites according to institutional protocol.			
• Cleanse skin using chlorhexidine shower or disposable wipes.			
• Maintain NPO status for at least 8 hours prior to the procedure			
• Provide patient education regarding medications, risk factors, peri-procedural/post-procedural care			
• Administer medications as ordered (antiplatelet, lipid lowering, antihypertensives excluding diuretics). Withhold oral hypoglycemic agents the morning of the procedure. Administer insulin based upon institutional protocol. Withhold antithrombotic medications as specified.			
• Assist surgeon as indicated in surgical site marking.			
• Perform pre-procedure verification process according to institutional protocol.			
• Administer antibiotic as ordered within one (1) hour of incision.			
Intra-Operative	**Yes**	**No**	**NA**
• Assemble surgical instruments per MD preference.			
• Attach ECG monitor, non-invasive BP cuff, SpO_2 probe to patient. Obtain baseline BP, heart rate, respirations, SpO_2, ECG rhythm.			
• Assist anesthesia as indicated with setup and insertion of arterial line.			
• Assist with positioning of head and neck.			
• Monitor vital signs, ECG rhythm and complications during procedure. Notify MD if present.			
• Administer ordered medications as indicated (unfractionated heparin, antihypertensives)			
Post-Operative	**Yes**	**No**	**NA**
• Monitor vital signs, ECG rhythm, pain and sedation levels at specified intervals. Include assessment of neck dressing & incision for hematoma or drainage (including bulb drain if present) with assessments. Notify MD of vital sign changes or neck hematoma develops or bulb drainage increases.			
• Perform neurological & cranial nerve assessment upon arrival and at scheduled intervals (orientation, Glasgow Coma Scale or other stroke scale as specified, pupil reaction)			
• Titrate IV antihypertensive medication to targeted systolic or mean arterial blood pressure			
• Titrate oxygen if ordered to maintain a SpO_2 of 94% in acutely ill patients & 92% in patients at risk for hypercapnic respiratory failure			
• Maintain patent IV site and continuous ECG monitoring until discharge or as specified by the MD			
• Obtain a 12-lead ECG at specified time			
• Remove any neck incision dressings at time interval specified. Maintain vacuum on bulb drain if present.			
• Keep the head of the bed at 30 degrees & maintain bed-rest for specified time interval			
• Resume clear liquids & progress diet when ordered. Assess for complaints of dysphagia & notify MD if present.			
• Assess for post-procedure complications – notify MD immediately if any occur			
• Provide patient education regarding risk factor modification, medications, activity restrictions, incision care, signs and symptoms to report to MD			
• Administer post-procedure medications (antiplatelet agents, antihypertensives). Follow institutional protocol for oral antithrombotics. If diabetic: hypoglycemics may be restarted the morning after the surgery. If angiographic confirmation of revascularization performed during the surgery, withhold metformin/derivatives for 48-hours after the procedure.			
• Schedule follow up completion imaging studies (such as carotid duplex ultrasound) as ordered			
• Assist in data collection/outcome reporting as indicated based upon institutional requirements			

**These are general recommendations – follow your institutional protocol

APPENDIX B
PERTINENT CRANIAL NERVE ASSESSMENT

Facial (VII): Test patient's ability to raise eyebrows, close eyelids, frown, smile, pucker, and taste. Symptoms of injury would include ipsilateral lip droop and inability to smile.

Glossopharyngeal (IX): Observe soft palate in the back of the upper mouth for equal movement and uvula for deviation. Check gag reflex. Symptoms of injury would include dysphagia with ipsilateral Horner syndrome (ptosis, exophthalmos, and decreased sweating).

Superior laryngeal vagus (X): Check speech and ability to swallow. Symptoms of injury may include a weak voice and dysphagia. Patient may be more prone to aspiration. Because the vagus nerve lies near the carotid artery, it may be affected by edema. As a result, bradycardia can occur, which can decrease cerebral perfusion and cause neurologic changes. In the period of 1–5 hours post procedurally, reflex hypotension can occur associated with hypovolemia or vagal stimulation.

Recurrent laryngeal-anterior vagus (X): Test swallowing and gag reflex. Symptoms of injury may present as vocal cord paralysis or inadequate gag reflex. Injury to more than one laryngeal nerve is associated with increased chance of stridor and airway obstruction.

Spinal Accessory (XI): Test patient's ability to move head laterally, flex neck and shrug shoulders. Symptoms of injury may affect neck and shoulder movement and alignment.

Hypoglossal (XII): Test the patient's tongue strength and ability to protrude tongue. Symptoms of injury are seen in the ipsilateral tongue, causing problems with speech and mastication. Unilateral motor damage to this nerve causes tongue deviation to the same side.

Figure 1
Arteries of the Head and Neck
(Figure published in: Moser, D., Reigel, B. (Eds), Cardiac Nursing, Figure 74-4, ©Elsevier 2007)

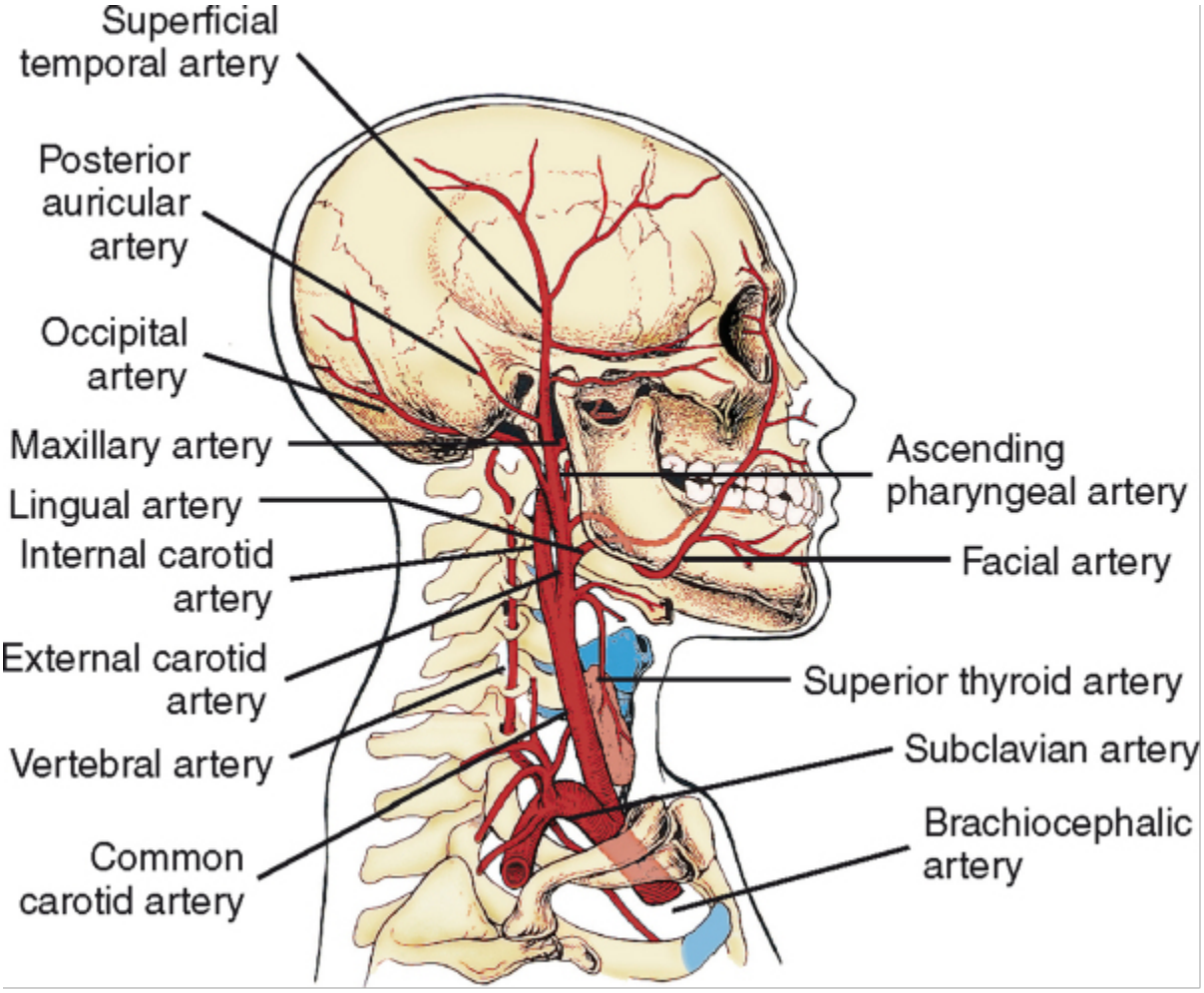

Figure 2
Carotid Artery with Atherosclerosis

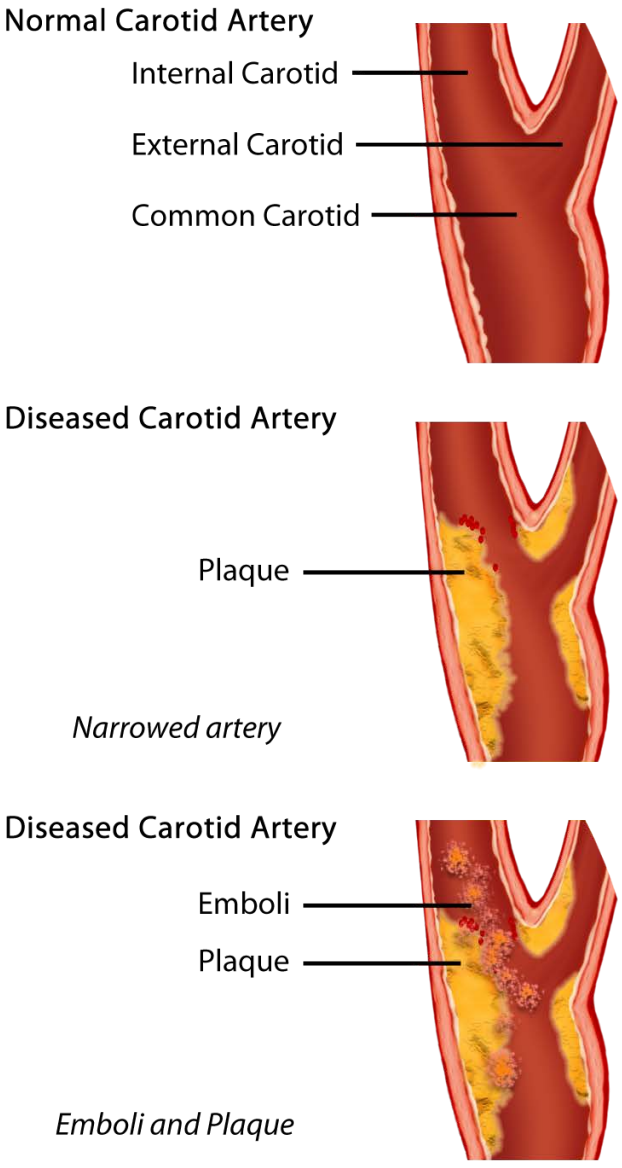

Figure 3
Carotid Endarterectomy Neck Incision Options