Treatment of Symptomatic Carotid Stenosis

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EPIDEMIOLOGY OF CAROTID DISEASE AND STROKE

The percentage of ischemic strokes resulting from atherosclerotic debris that embolizes from the extracranial carotid artery into the cerebral circulation is 20% to 30%.[1,2] Atherosclerosis results from the adverse impact of modifiable risk factors, resulting in inflammation within the circulatory system that causes endothelial injury whose end result is calcium deposition within the arterial wall. These risk factors for developing carotid atherosclerotic disease include smoking, hypertension, hypercholesterolemia, and diabetes.

Smoking is well established to be strongly associated with the development of carotid atherosclerotic disease. In the comparison of age-matched non-smokers, former smokers, and current smokers, the prevalence of clinically significant carotid disease (>50%) was seen in 4.4%, 7.3%, and 9.5% ($p < 0.0001$), respectively.[3] For every 20 mm Hg increase in systolic blood pressure, the odds ratio of developing moderate carotid stenosis is 2.11. Additionally, every 10 mg/dL increase in serum cholesterol level was associated with an odds ratio of 1.10 for developing hemodynamically significant carotid stenosis.[3]

The prevalence of carotid stenosis varies with geographic location due to cultural, genetic, and socioeconomic differences. The southeastern part of the United States has been coined the "stroke belt" because adjusted stroke rates have been shown to be 10% higher than the national average.[4] Nevertheless, the contribution of carotid artery disease to this increased stroke incidence has not been well defined.[5]

CAROTID ARTERY IMAGING IN THE DIAGNOSIS OF CAROTID DISEASE

The diagnostic tools used to image the carotid arteries are duplex ultrasonography (DUS), computed tomography angiography (CTA), magnetic resonance angiography (MRA), and digital subtraction angiography (DSA). The majority of vascular surgeons perform carotid surgery solely from the information provided by the DUS if the performing lab has demonstrated duplex accuracy commensurate with the accrediting bodies that oversee noninvasive vascular laboratories. In the vast majority of cases, this is reasonable and appropriate, and further imaging would not add useful information to the treatment algorithm. In certain instances of discordant information (e.g., significant visualized internal carotid stenosis without velocity elevation or global velocity reductions), or if the planned procedure is carotid stenting, a noninvasive imaging procedure should be performed—either CTA or MRA, depending upon the institution-specific accuracy of the respective modality. Although invasive, DSA remains the gold standard for carotid imaging, but it should rarely be required solely for diagnostic purposes. Prior to carotid stenting, CTA and MRA allow assessment of the carotid artery from the aortic arch to the carotid siphon and intracranially and thus are recommended to be performed prior to all procedures.

CAROTID REVASCULARIZATION FOR STROKE PREVENTION

Carotid Endarterectomy

Carotid endarterectomy (CEA) is arguably the most successful and most rigorously studied surgical procedure in the history of American surgery. First performed in the 1950s, it ranks as one of the most commonly performed peripheral arterial procedures performed in the United States. The initial success in surgical revascularization of the carotid bifurcation was performed by Carrera, Eastcott, Pickering, and Robb in 1954. Controversially, DeBakey reported that he performed the first CEA in 1953, yet it was not published at the time.[6] In the aftermath of several early clinic trials, CEA became extremely popular, despite criticism from the neurology community, because superiority of surgery over medical therapy had not been definitively established. Rates of CEA appropriately diminished throughout the vascular community after the publication of several studies demonstrating that the rates of complications of carotid endarterectomy were excessive.[7] These concerns resulted in the design and execution of several landmark randomized trials in the 1990s under an independent neurological audit that established the role of CEA versus medical treatment for stroke prevention in symptomatic patients (e.g., those presenting with carotid

stenosis and an ipsilateral stroke, transient ischemic attack [TIA] or amarosis fugax). These trials—primarily, North American Symptomatic Carotid Endarterectomy Trial (NASCET) and European Carotid Surgery Trial (ECST)—showing benefit from carotid endarterectomy were associated with a dramatic resurgence in the rates of the procedure.

The ECST and NASCET trials were the first to show that CEA was highly beneficial in those with ≥70% stenosis (absolute risk reduction of 16.0%, $p < 0.001$) and a slight but significant benefit in those with 50% to 69% stenosis (absolute risk reduction of 4.6%, $p = 0.04$); the results are summarized in Table 12.1.[8] Critically, the benefit in stroke reduction from CEA relates to the complication rate of the procedure. Reported benefits for NASCET and ECST were predicated on the perioperative risks of stroke or death of 7.5% in the ECST and 6.5% in the NASCET. If major stroke and death rates exceed this by any degree, all benefit from carotid revascularization with CEA is lost.[9] The best outcome to date regarding the efficacy of CEA in symptomatic patients was seen in the Carotid Revascularization Endarterectomy Versus Stent Trial (CREST), where at 30 days, the rate of stroke and death rate was 3.2%.[10]

Despite these admirable results for CEA in the symptomatic cohort, a major criticism centered on how the rigorous selection of patients included in these trials did not represent a "real-world" population of those individuals largely undergoing CEA. The coexistence of coronary artery disease and/or congestive heart failure (CHF) increases mortality after any vascular surgical procedure.

TABLE 12.1 Comparison of the results of the ECST and NASCET trials stratified by degree of stenosis for symptomatic patients

Degree stenosis	Number of patients	Medical risk (%) at 2 years	Surgical risk (%) at 2 years	Risk difference (%)	Relative risk reduction (%)	No need to treat*	Perioperative stroke and death rate (%)
70%–99% NASCET	659	21.4	8.6	12.8	60	8	5.8
70%–99% ECST	501	19.9	7.0	12.9	65	8	5.6
50%–69% NASCET	858	14.2	9.2	5.0	35	20	7.1
50%–69% ECST	684	9.7	11.1	–1.4	–14	—	9.8
<50% NASCET	1368	11.6	10.1	1.5	13	67	6.5
<50% ECST	1882	4.3	9.5	–5.2	–109	—	6.1

Source: Adapted from Reference 8.

The aforementioned trials excluded patients with unstable angina, myocardial infarction in the prior 6 months, CHF, and active coronary disease requiring revascularization.[11] Other factors resulting in exclusion from these CEA trials centered on factors related to the actual surgical site: prior endarterectomy, prior neck dissection or radiation, and surgically inaccessible high or low lesions. Patients with contralateral carotid occlusions were also thought to be problematic because they were felt to have an increased the risk of stroke with CEA. Individuals with one or more of these commonly present conditions would have been denied entry into trial; thus, information regarding the outcome with carotid revascularization via CEA in this "high-risk" population was lacking, resulting in the evolution of the less invasive, non-surgical technique of carotid angioplasty and stenting (CAS).[12]

Carotid Angioplasty and Stenting

First performed by Matthias in 1994, CAS as treatment for carotid stenosis has witnessed a historical progression very similar to CEA, with subsequent trials producing improved results as more knowledge is acquired regarding patient selection and as technological improvements are made (see Figure 12.1). Although currently approved by the U.S. Food and Drug Administration (FDA) for treatment of moderate-risk patients with carotid disease, the Centers for Medicare and Medicaid Services (CMS) has maintained approval of reimbursement only for those individuals with symptomatic, >70% lesions with a "high-risk" qualifying condition. The high-risk criteria are Class III/IV congestive heart failure, left ventricular ejection fraction <30%, open heart surgery within 6 weeks, recent myocardial infarction (>24 hours to <30 days), unstable angina: Class III/IV, concurrent requirement for coronary revascularization, severe pulmonary disease, contralateral carotid occlusion, previous radiation to head/neck, previous CEA, age >80 years, and surgically inaccessible lesions.[13]

The primary limitation for performing CAS is unfavorable anatomy.[14] Unfavorable aortic arch types, vascular anomalies such as bovine anatomy, proximal and distal tortuosity, and other specific arterial lesions can reduce success with this approach, leading to an increase in adverse outcomes.[15,16] Although most CEAs are performed with duplex ultrasound alone, pre-procedure imaging such as CTA or MRA should be performed on all patients being considered for CAS to establish favorable anatomy prior to invasive angiography, to improve patient selection, and to avoid the risks of angiography in those who are poor candidates for CAS.

The initial major trials regarding the efficacy of carotid stenting were performed in a patient population that would not have met the entry criteria for NASCET or ECST due to one of the preceding disqualifying conditions. Both

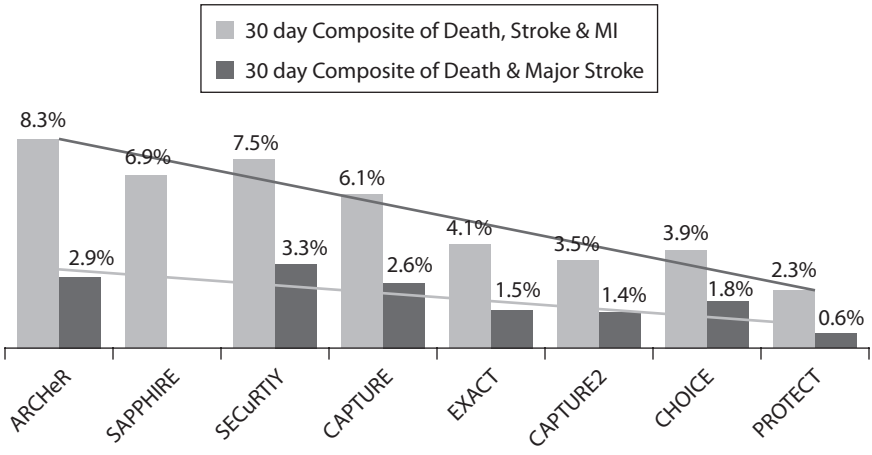

FIGURE 12.1 Carotid stenting outcome improvement over time

Source: Adapted from Dr. Thomas Brott and the CREST investigators.

the ACCULINK for Revascularization of Carotids in High Risk Patient (ARCHER) and the Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy (SAPPHIRE) trials established carotid stenting as a feasible revascularization modality for carotid disease in a selected minority of patients deemed high risk for CEA. The two major studies comparing CEA and CAS in the low- to moderate-risk population are the International Carotid Stenting Study (ICSS) and the CREST trial. Both trials compared carotid endarterectomy with stenting in patients eligible for either procedure.

ICSS was a multicenter, international, randomized controlled trial comparing carotid artery stenting with carotid endarterectomy in patients with recently symptomatic carotid stenosis; the results are summarized in Table 12.2.[8] The trial enrolled 1,713 patients, with 855 randomized to stenting and 858 randomized to surgery. The incidence of stroke, death, or procedural myocardial infarction was 8.5% in the stenting group compared with 5.2% in the endarterectomy group (HR 1.69, 1.16–2.45, $p=0.006$). Risks of any stroke and all-cause death were higher in the stenting group than in the endarterectomy group. The difference was driven largely by minor strokes yet was offset by a higher frequency of cranial nerve palsy with endarterectomy. The authors concluded that carotid endarterectomy should be the treatment of choice for suitable patients with recently symptomatic carotid artery stenosis. Although ICSS concluded that endarterectomy should be the treatment of choice, there was an inference that some individuals may be better suited for carotid stenting. This inference was substantiated and supported by CREST.[10]

TABLE 12.2 ICSS: 120-day interim safety results

Endpoint	Stenting group, number (%)	Carotid endarterectomy group, number (%)	Hazard ratio (95% CI)	*p*-Value
Disabling stroke or death	34 (4.0)	27 (3.2)	1.28 (0.77–2.11)	0.34
Stroke, death, or procedural MI	72 (8.5)	44 (5.2)	1.69 (1.16–2.45)	0.006
Any stroke	65 (7.7)	35 (4.1)	1.92 (1.27–2.89)	0.002
All-cause death	19 (2.3)	7 (0.8)	2.76 (1.16–6.56)	0.017

Source: Adapted from Reference 8.

Note: CI = confidence interval; ICSS = International Carotid Stenting Study; MI = myocardial infarction

CREST was a multicenter trial supported by the National Institutes of Health. The study included symptomatic (>50% stenosis) and asymptomatic (>70% stenosis) patients. As the largest study on carotid revascularization, which enrolled 2,500 patients from 126 sites throughout North America, a major hallmark of CREST was the most rigorous operator/surgeon entry criteria to date. Sites could not enroll in the trial until the operators performing carotid artery stenting and carotid endarterectomy had been approved and certified by the interventional and surgical management committees, respectively. Certification was achieved by 477 surgeons, whose clinical outcomes were audited by means of a detailed, rigorous selection process documenting that they performed more than 12 procedures per year with rates of complications and death less than 3% among asymptomatic patients and less than 5% among symptomatic patients. A total of 225 interventionists were approved after satisfactory evaluation of their endovascular experience and carotid-stenting results, participation in hands-on training, and participation in a lead-in phase of training.[10] All centers were required to have a team consisting of a neurologist, a surgeon, and an interventionist.

The results demonstrated no significant differences with regard to the combined primary endpoints of stroke, death, and myocardial infarction (MI) with CAS or CEA (7.2% vs. 6.8%, $p = 0.51$) (see Table 12.3). Notwithstanding, periprocedural strokes in the CAS group were significantly greater than the CEA group (4.1 vs. 2.3, $p = 0.01$), yet this was at the expense of a greater number of MIs in the CEA group compared to the CAS group (2.3 vs. 1.1, $p = 0.03$). Quality-of-life assessment data performed indicated a significant negative impact was associated with periprocedural stroke rather than with MI.[17]

TABLE 12.3 CREST primary endpoint results (stroke/MI/death)

CAS	7.2%
CEA	6.8%
Hazard ratio	1.11
95% CI	0.81–1.51
p-value	0.51

Cranial nerve injury occurred in 4.6% of those undergoing CEA, but residual effects were largely nonexistent at 1 year.[18]

Another important finding from CREST was derived from the hazard ratios for the primary endpoint, as calculated for the CAS group versus the CEA group according to age at the time of the procedure. These ratios were estimated from the proportional-hazards model with adjustment for sex and symptomatic status. Graphing of these data revealed that CAS is likely safer in the younger population and that CEA is likely safer in the older patient, with roughly 70 years representing that age demarcation (Figure 12.2).[10] This mirrors the clinical realm where older patients are more likely to have both elongation and a higher calcified plaque burden of the thoracic aortic arch.[19] Additionally, elderly patients have a reduced cerebral vascular reserve, rendering them more susceptible to the effects of embolization.[20]

This important finding of the cohort likely to perform better with the respective procedure helped to clarify the inference established in the ICSS trial. The results of CREST were palatable for both the surgical and intervention communities. Surgeons were encouraged that CEA resulted in lower stroke rates, and interventionists were content that the primary endpoint (stroke/death/MI) was similar between the two methods.

Although carotid revascularization for symptomatic, >50% lesions is not debatable, significant discord exists regarding the treatment of asymptomatic, noncritical carotid lesions. With data extrapolated from randomized trials assessing treatment for intracranial arterial stenosis,[21,22] some may argue that contemporary pharmacologic treatments (including intensively monitored treatments for hypertension, hyperlipidemia, diabetes, and smoking cessation) are acceptable in stroke reduction in those with asymptomatic moderate carotid disease.

There has been no adequately powered assessment of "modern-day" medical management of asymptomatic carotid disease since the ACAS trial 2 decades ago. CREST-2 will assess CEA and CAS, respectively, to the best medical therapy

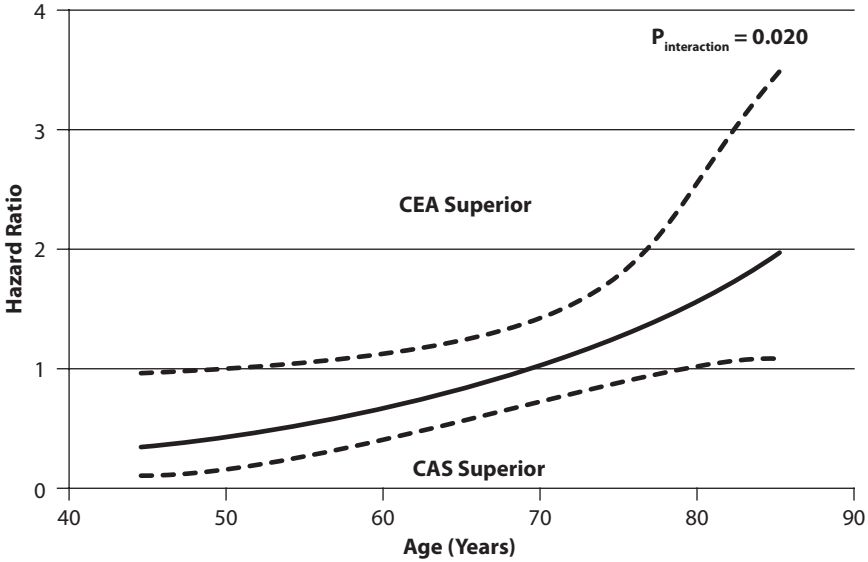

FIGURE 12.2 CREST 4-year primary outcomes

in the asymptomatic cohort. The primary objectives of the trial are (a) to determine if intensive medical therapy alone (mirrored after the aggressive medical management arm of the SAMMPRIS trial, reviewed in Chapter 13) is different from CAS plus intensive medical therapy and (b) to determine if intensive medical therapy is different from CEA plus intensive medical therapy. The primary endpoint will be any stroke or death during the periprocedural period and ipsilateral stroke thereafter, out to 4 years of follow-up. Eligibility criteria include asymptomatic status for less than 180 days from the time of the baseline assessment, carotid stenosis ≥70% as determined by duplex ultrasound, and one confirmatory study (MR or CT angiography). Patients will be randomized to only one of two trials within CREST-2: either CAS plus medical management compared to medical management alone or CEA plus medical management compared to medical management alone.[23] Notably, 50% of patients will be randomized to intensive medical therapy.

COMPLICATIONS OF CEA

Stroke

Neurological complications after CEA are one of the most devastating postoperative complications in the entire field of surgery—particularly if the indication for endarterectomy is asymptomatic disease. An important factor paramount

to acceptably low postoperative stroke rates is the CEA case volume of the surgeon.[24–26]

Surgical factors contributing to postoperative neurological events are plaque embolism, carotid occlusion due to platelet aggregation, and inadequate cerebral protection intraoperatively. Trailing only MI, stroke is the second most common cause of death following CEA. Acceptable postoperative stroke rates are <3% for asymptomatic patients and 5% to 7% for symptomatic patients. Any neurologic change in the patient after CEA is a technical problem at the endarterectomy site, until proven otherwise, and warrants expeditious return to the operating room (OR) for evaluation and angiography. If no technical issue is present at reexploration and angiography reveals embolic occlusion of an intracerebral artery, neuro-rescue techniques with intracranial thrombolysis and/or plaque or clot retrieval devices may be employed, if available.

Carotid artery stenting may also be effective for managing perioperative stroke after CEA. In the setting of an anatomically high lesion that proved technically difficult surgically, carotid stenting may provide an adjunctive method to address a flow-limiting lesion that was difficult to address intraoperatively.[27] Carotid stenting, however, is not considered standard for treatment of acute complications of carotid endarterectomy. Because the majority of postoperative events occur in the immediate time period after CEA, our convention is to have all post-op CEA patients remain in the recovery room for 2 hours prior to disposition to either the surgical ward or the intensive care unit. This affords both frequent and continuous neurological assessment by the same attendant and the ability to quickly return to the OR for reexploration if necessary. If there are no blood pressure issues postoperatively, we disposition patients to the surgical floor with neurological and vital assessments every 2 hours with planned discharge for the following day. If there are any hyper- or hypotension issues requiring continuous intravenous pharmacological manipulation, we maintain the arterial line monitoring and disposition patients to the intensive care unit for observation.

Cranial Nerve Injury

Cranial nerve injuries occur in roughly 5% of patients following CEA, with the vast majority resolving completely by 6 months post-surgery.[26–28] This was corroborated in both the ECST and CREST trials. In ECST, the rate of cranial nerve injury postoperatively was 5.1% with resolution of slightly less than half of these injuries by hospital discharge.[28] Typically, the hypoglossal nerve was most frequently involved, occurring in 3% post-CEA, followed by the marginal mandibular branch of the facial nerve at 2%. Vagus and glossopharyngeal nerve injuries occur rarely because they are not encountered in a clinically

relevant manner during CEA dissection, yet they may occur at a higher rate during difficult exposures. Factors that increase the risk of cranial nerve injury include urgent procedures, immediate reexploration, and return to the OR for a neurologic event or bleeding. Interestingly, and contrary to conventional teachings, redoing CEA or prior cervical radiation were *not* associated with an increased risk.[26]

Cerebral Hyperperfusion Syndrome

The cerebral hyperperfusion syndrome (CHS) can be a devastating post-carotid revascularization complication that occurs in a small percentage of patients (0.05%–3%). CHS can result in intracerebral hemorrhage post–carotid revascularization and is associated with typical clinical hallmarks[28–34] (see Table 12.4). Despite an unclear mechanism, it is thought that the cause is related to loss of intracerebral autoregulation of perfusion in the previously ischemic carotid vascular bed. To maintain sufficient cerebral blood flow, small vessels compensate with chronic arteriolar maximal dilatation. After correction of the carotid stenosis, blood flow is restored to a normal or elevated perfusion pressure within the previously hypoperfused hemisphere. The dilated vessels are thought to be unable to assume normal tone or to vasoconstrict sufficiently to protect the capillary bed from excess flow, resulting in edema and hemorrhage, which in turn results in the clinical manifestations. Besides the initial presence of critical unilateral or bilateral carotid lesions, post-revascularization hypertension frequently is an associated predecessor of the syndrome, underscoring the importance of good perioperative blood pressure control.[8] Some evidence suggests that this syndrome may be more likely when revascularization is performed after recent stroke.[35–37] This is particularly important because contemporary practitioners now tend to perform post-stroke carotid revascularization without delay due to data refuting the previous practice of a several-week delay.[38,39]

TABLE 12.4 Hyperperfusion features

- Headache ipsilateral to the revascularized internal carotid, typically improved in upright posture; may herald the syndrome in the first week after endarterectomy.
- Focal motor seizures are common, sometimes with post-ictal Todd's paralysis mimicking post-endarterectomy stroke from carotid thrombosis.
- Intracerebral hemorrhage is the most feared complication, occurring in about 0.6% of patients after CEA, usually within 2 weeks of surgery.

Source: Adapted from Reference 34.

The prevention of this complication is aided with fastidious control of postoperative hypertension. Blood pressure parameters depend on the initial severity of stenosis and presumed or known degree of capillary dilatation before surgery. For severe stenosis (>90%), our convention is to maintain systolic blood pressure below 150 mm Hg as aggressively as required with the liberal use of continuous-drip intravenous antihypertensives in short order if bolus intravenous methods fail. Theoretically, patients with less severe stenosis require less conservative parameters than those with severe stenosis; however, it may be more complicated than this because patients have varying sources and flow contribution from collaterals likely contributing to the cerebrovascular reserve. The vast majority of patients do not require sustained antihypertensive infusion and are restarted on their home oral regimen in the first 12 hours after the procedure. Any complaint of severe headache following revascularization is the syndrome, unless proven otherwise, and should be evaluated expeditiously with head CT. All antithrombotics should be discontinued if hemorrhage is confirmed, and platelet transfusion to reverse their effect should be considered. Seizures related to hyperperfusion are usually successfully treated with standard antiepileptic drugs.[39]

Restenosis

Restenosis of the carotid artery after CEA occurs in 3% to 10% of patients and underscores the importance of yearly duplex assessment, even though the majority have little indication for repeat revascularization.[40,41] It is also not uncommon to observe mild restenosis in the initial months after CEA that normalizes after a year, likely the result of carotid remodeling. Although not definitively proven, statin drugs may be protective against restenosis.[42] Additionally, patch angioplasty during CEA has been associated with a decreased risk of long-term recurrent stenosis compared with primary closure.[43]

The mechanism of the restenotic lesion has a direct relationship to the time point of presentation after initial surgery.[44,45] Restenosis occurring within 2 to 3 years after CEA is thought to be attributed to intimal hyperplasia that creates a smooth, tapered lesion with a low likelihood of embolization. Conversely, restenosis after 2 to 3 years is thought to be from the progression of atherosclerotic disease process and thus presents as an irregular plaque that may serve as an embolic source. The former typically does not require intervention unless a near-occlusive lesion results, whereas the latter requires correction if symptoms develop in association with critical restenosis (>70%). Restenosis of post-CEA carotid lesions is increasingly (and appropriately) treated via carotid stenting, when feasible, thus avoiding the potential risks inherent with redo surgical neck exploration.

COMPLICATIONS OF CAROTID STENTING

Carotid artery stenting (CAS) has certain advantages compared to CEA, primarily as it pertains to the avoidance of both an incision and general anesthesia. Resultantly cranial nerve injuries, neck hematomas, and anesthetic/intubation complications are not encountered. However, similar complications of stroke, including cerebral hyperperfusion syndrome and restenosis, likewise occur to some degree with CAS. Additionally, CAS has the unique complications related to access not present in those undergoing CEA.

Stroke

The most serious acute complication associated with CAS is stroke. Periprocedural stroke may develop from several mechanisms: thromboembolism, hemodynamic alteration–induced hypoperfusion, cerebral hyperperfusion, and intracerebral hemorrhage. In addition, filter embolic protection devices—the most common device type used currently—may result in an angiographic "slow-flow phenomenon," appearing as reduced or absent anterograde flow in the internal carotid artery caused by occlusion of the filter membrane pores by microemboli and debris. Occurring in 9% of cases in one study, patients with the slow-flow phenomenon had an increased 30-day risk of stroke compared with those who did not (9.5% vs. 1.7%).[46] Protection devices may also cause arterial spasm and dissection that may lead to periprocedural cerebral events.

Carotid plaque morphology and characteristics also contribute to increased stroke rates with carotid stenting. Ulcerated carotid plaque, increasing degree of carotid stenosis, and longer carotid lesions are aspects of carotid disease associated with an increased risk for stroke.[47] Retrospective studies also suggest that long carotid lesions (>10 mm) or tandem carotid lesions with more than one lesion separated by normal vessel wall have also been associated with a higher stroke risk.[48,49] Appropriate patient selection is the most important factor in minimizing post-CAS stroke. Adequate pre-procedural planning that assesses arch anatomy, carotid tortuosity, and lesion characteristics can minimize the occurrence of this potentially devastating complication.

Hemodynamic Instability

Hemodynamic lability, such as bradycardia and hypotension, occurs frequently after carotid artery stenting; however, associated morbidity has not been established. Occurring in an estimated 20% to 30% of cases, in most patients post-procedural hypotension resolves within 12 hours.[50] The clinical manifestations are thought to be due to the effects of balloon inflation on the

carotid baroreceptors during CAS. Carotid sinus baroreceptors are located within the adventitia of the origin of the internal carotid artery and are innervated by a branch of the glossopharyngeal nerve (the sinus nerve of Hering). In response to low blood pressure, the nerve fibers decrease their firing rates, stimulating the sympathetic nervous system and inhibiting the parasympathetic nervous system via a centrally acting mechanism. Hemodynamic instability following CAS is significantly associated with age, >10-mm distance between the carotid bifurcation and the site of minimum lumen diameter, and prior ipsilateral carotid endarterectomy and recent stroke.[50] Neither hypotension nor bradycardia has been predictive of myocardial infarction, stroke, or death.[51]

Access-Related Complications

Access-related issues are grouped into two categories: complications involving the arterial puncture or due to distal embolization. Arterial puncture complication includes hematoma and pseudoaneurysm formation. Distal embolization complications may be due to atheroembolization as a result of a diseased femoral artery or closure device misadventure. Arterial puncture issues may be nearly completely eliminated if care is taken to puncture the common femoral artery in a fluoroscopically confirmed segment overlying the femoral head. The risk factors for pseudoaneurysm development include inadequate post-procedure compression of the puncture site, post-procedural anticoagulation, antiplatelet therapy during the intervention, age >65 years, obesity, hypertension, peripheral artery disease, and hemodialysis.[52]

During any transfemoral catheterization procedure, plaque may become dislodged either from a diseased femoral artery or from more proximal aortic or iliac disease, with occasional limb-threatening ischemia in some instances. Care should be taken to ensure that the targeted femoral artery is suitable for puncture. Fluoroscopic visualization prior to arterial puncture can often demonstrate plaque burden, leading to selection of the contralateral femoral site for puncture.

Restenosis

Restenosis rates after CAS vary, yet overall the durability compares favorably to CEA.[53–55] Meta-analysis of multiple studies shows that restenosis occurs in about 6% of arteries after 1 year. Historical restenosis rates for CEA ranges in the 5% to 10% range.[53] CREST reported no difference in restenosis at 24 months for carotid artery stenting compared with carotid endarterectomy.[10] Secondary analysis revealed that female sex, dyslipidemia, and diabetes were independent predictors of restenosis for both procedures. Interestingly, smoking increased the CEA rate of restenosis but not after CAS.

CONCLUSION

In summary, carotid disease's contribution to stroke remains significant. Despite advances in the surgical and interventional approaches to treatment, carotid disease–related stroke continues to remain an important source of morbidity and mortality. Carotid endarterectomy remains the gold standard for treatment of significant disease. Carotid angioplasty and stenting has been a welcomed addition to the treatment armamentarium; however, there remain strict guidelines for patient inclusion and significant obstacles for practitioners to obtain and maintain expertise. Current studies are under way to assess the efficacy of pharmacologic agents in the treatment of carotid disease, which will provide valuable information as we continue to improve our total approach for those afflicted.

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