Management of Asymptomatic Internal Carotid Artery Stenosis

Joshua A. Beckman, MD

Patient Presentation

Mrs H is a 78-year-old woman with a 15-year history of hypertension and a 25 pack-year smoking history (last cigarette smoked in 2004) who was referred by her primary care physician for carotid ultrasonography because the physician noted a right carotid artery bruit. The patient was found to have an 80% to 99% right internal carotid artery (ICA) stenosis (Figure). The patient denies any symptoms related to her carotid artery stenosis, including dysarthria, ataxia, weakness, or paresthesia. The patient is able to perform her activities of daily living, participates in activities several days per week at the local senior center, and walks for 20 minutes 3 times a week. Her medical therapy includes lisinopril, hydrochlorothiazide, atorvastatin, and vitamin D. Other than the carotid bruit, her physical examination is unremarkable.

The patient asked her physician the following questions: What is my risk of stroke? What is the best way to treat it? and How risky are the procedures to treat the artery narrowing?

Discussion

Asymptomatic ICA stenosis is a common problem in older patients, developing in 7% to 9% of patients by age 75 years.1-3 Moreover, 1% to 2% of patients aged between 65 and 84 years will have sufficiently severe asymptomatic ICA stenosis to warrant consideration of carotid artery revascularization.2,4 Primary care clinicians will frequently encounter patients with asymptomatic ICA stenosis and must decide whether to treat the patient with medical therapy alone or refer the patient for surgical or percutaneous revascularization.

The care of the patient with asymptomatic ICA stenosis may be influenced by referral patterns, access to surgical therapy, and the clinician’s preference for percutaneous or medical or surgical therapy.5 Challenges associated with decision making for patients with asymptomatic ICA stenosis arise from the evolution of all facets of the disease, including reduced risk from revascularization procedures; improved medical therapy, which may lower the risk of stroke without revascularization; and overall decreasing cardiovascular mortality.6-8 These recent trends have raised questions about the applicability of prior randomized trials of carotid endarterectomy (CEA) to patients with asymptomatic ICA stenosis in current medical practice.

Risk of Stroke

This patient case highlights the challenges in caring for patients with asymptomatic ICA stenosis in identifying the optimal therapeutic course given the low risk of stroke, the potentially catastrophic consequences of a major stroke, and the risks of carotid artery revascularization. Little agreement exists in the medical community about the best approach in a patient similar to this one. Practice patterns for asymptomatic ICA stenosis are widely variable and range from infrequent use of revascularization9 to routine use of only surgical revascularization in patients at standard risk.10 Multispecialty guidelines on extracranial carotid disease state that CEA is reasonable “in asymptomatic patients who have more than 70% stenosis of the internal carotid artery if the risk of perioperative stroke, MI [myocar-
The guidelines further recommend consideration of prophylactic carotid artery stenting (CAS) in highly selected patients with asymptomatic ICA stenosis (minimum 60% by angiography, 70% by validated Doppler ultrasound), but state that the efficacy of prophylactic CAS compared with medical therapy alone is not well established for these patients. To appropriately counsel patients with asymptomatic ICA stenosis requires knowledge of the current stroke risk associated with asymptomatic ICA stenosis and knowledge of secular trends in asymptomatic ICA stenosis associated with medical management and revascularization.

Internal carotid artery stenosis is associated with approximately 10% to 15% of ischemic stroke events in the United States; however, cryptogenic stroke and cardioembolic stroke are much more common. Concerns about the potential catastrophic functional outcomes after stroke are an important factor in clinical decision making for patients with asymptomatic carotid artery disease. In contrast, mortality and functional outcomes after MI have significantly improved in recent decades. For example, an hospital admission for acute coronary syndrome is typically approximately 48 hours in duration with resumption of activities and work soon thereafter. In contrast, concern regarding stroke and its potential for devastating outcomes remains high. Within the Framingham study data, patients aged 60 years had an expected lifespan of 20 more years. A patient who had a stroke had a reduced life expectancy by 12 years. However, this shortened life expectancy is less worrisome than the severe disability that may occur with major stroke.

Despite the potential for catastrophic outcomes after a major stroke, risks associated with asymptomatic ICA disease have decreased over time. In the 1980s, the risk of stroke for individuals with an 80% to 99% stenosis was estimated at approximately 3% per year and the risk of stroke for individuals with a 90% to 99% stenosis was estimated at approximately 5% per year. However, these rates have significantly declined in the past 30 years. Recent data show that rates of stroke associated with asymptomatic ICA disease are only 0.5% to 1.0% per year in patients with a carotid stenosis of more than 50%. Rates of stroke diminished substantially after 2002, when the Heart Protection Study (HPS) was published. This study demonstrated the benefits of high-dose simvastatin for preventing stroke among patients at high risk of cardiovascular events. A recent meta-regression analysis of 30 studies of patients with asymptomatic ICA disease demonstrated that stroke rates have decreased from 2.83% before 2000 to 1.13% per year after 2000.

During the past 2 to 3 decades, the prevalence of ischemic heart disease has decreased by more than 30%, the rate of smoking has decreased by 14%, the incidence of atrial fibrillation has decreased by 6%, and the rate of peripheral artery disease may have decreased by 6%. These trends are associated with lower rates of stroke. For example, in the Asymptomatic Carotid Surgery Trial (ACST), the rate of ipsilateral stroke in the last 5 years of the trial was approximately 0.7% per year compared with 1.1% during the first 5 years of the trial and 2.2% during the 5 years of the Asymptomatic Carotid Atherosclerosis Study (ACAS).

Medical Therapy for Patients With Asymptomatic ICA Stenosis
The marked increase in prescription of effective therapies for cardiovascular risk reduction during the last decade has occurred simultaneously with a significant improvement in the natural history of carotid atherosclerosis. Of the many risk-reduction therapies, the most contentious one is aspirin. In the primary prevention setting,

![Figure. Mrs H's Doppler Ultrasound of the Right Internal Carotid Artery](image-url)
little data exist demonstrating any benefit in stroke reduction. The strongest data came from the Women’s Health Study,19 in which aspirin did not reduce the primary end point of nonfatal MI, nonfatal stroke, and death from cardiovascular causes. In a secondary end point, 1.1% of women allocated to the aspirin group developed a stroke compared with 1.3% of women allocated to the placebo group during the 10 years of follow-up. However, this difference was not statistically significant. The end point in the Women’s Health Study was for all participants, not for women with carotid artery stenosis.

In the Asymptomatic Cervical Bruit Study,20,26 the largest trial to directly test the benefit of aspirin in individuals with asymptomatic carotid stenosis, 372 patients with at least 50% carotid stenosis identified by duplex ultrasonography were randomly allocated to and administered aspirin (325 mg/d) or placebo. Patients were excluded for symptomatic cerebrovascular disease, valvular heart disease, atrial fibrillation, recent cardiovascular events, and a life expectancy of less than 5 years. There was no difference in the stroke rates between the 2 groups. In addition, in 1992, the Mayo Clinic Asymptomatic Carotid Endarterectomy study21 showed a reduction in MI in patients with asymptomatic ICA disease undergoing CEA and treated with aspirin. These trials are older and their applicability to the practice of medicine in 2013 and beyond is unclear.

In contrast with antiplatelet therapy, the association of blood pressure reduction with a lower incidence of stroke is clear. The benefit of blood pressure reduction in stroke prevention has been demonstrated for diuretics, calcium channel antagonists, β-blockers, and antagonists of the renin angiotensin system.22-24 In a small study of 332 patients with asymptomatic ICA stenosis with a mean follow-up of 44 months, higher blood pressure was associated with an increased risk of stroke.25 Antagonists of the renin angiotensin system may play a particularly important role in patients with cerebral vascular disease. In the Heart Outcomes Prevention Evaluation trial,26 patients with cerebrovascular disease had a 30% reduction in the rate of stroke during 4.5 years of follow-up. In the Losartan Intervention For Endpoint Reduction in Hypertension Study trial,27 losartan reduced the rate of fatal and nonfatal stroke by approximately 25% compared with treatment with atenolol in patients with essential hypertension and left ventricular hypertrophy.

Statin therapy may be largely responsible for the decrease in incidence of stroke in the past 10 years. Before the HPS, the association between cholesterol levels and stroke was modest, with no recommendation for statin therapy in patients with asymptomatic carotid disease.28 In the HPS, 20 536 patients aged between 40 and 80 years with a history of coronary disease, other occlusive arterial disease, or diabetes were randomized to 40 mg/d of simvastatin or matching placebo.7 Allocation to 40 mg/d of simvastatin was associated with a 25% reduction in the rate of stroke compared with placebo.7 The reductions in ischemic stroke were similar in patients with and without previous cerebrovascular disease.29 Furthermore, the addition of a statin compared with placebo reduced the rate of carotid revascularization by 50%.7 The value of statins more, the addition of a statin compared with placebo reduced the rate of carotid revascularization by 50%.7 The value of statins more, the addition of a statin compared with placebo reduced the rate of carotid revascularization by 50%.7 The value of statins more, the addition of a statin compared with placebo reduced the rate of carotid revascularization by 50%.7 The value of statins more, the addition of a statin compared with placebo reduced the rate of carotid revascularization by 50%.7 The value of statins more, the addition of a statin compared with placebo reduced the rate of carotid revascularization by 50%.7 The value of statins more, the addition of a statin compared with placebo reduced the rate of carotid revascularization by 50%.7 The value of statins more, the addition of a statin compared with placebo reduced the rate of carotid revascularization by 50%.7 The value of statins more, the addition of a statin compared with placebo reduced the rate of carotid revascularization by 50%.7 The value of statins more, the addition of a statin compared with placebo reduced the rate of carotid revascularization by 50%.7 The value of statins

**Revascularization for Asymptomatic ICA Stenosis**

Carotid revascularization represents the most controversial area in the management of asymptomatic ICA disease. Three level 1 studies provide most of the evidence that guides current therapy (Table 1).35-33 A level 1 study represents evidence obtained from a properly designed randomized control study as defined by the US Preventive Services Task Force.34 The first study providing this evidence is ACAS.31 A total of 1662 patients aged 40 to 79 years with asymptomatic ICA stenosis of 60% or more reduction in diameter were randomly allocated to CEA or medical therapy between December 1987 and December 1993. All patients received aspirin.31 Patients were excluded for recent cerebrovascular events, the likelihood of death or significant disability within 5 years, or the possibility of a cardiac embolism. With a median follow-up of 2.7 years, participants randomized to CEA had an estimated reduction over 5 years in the primary end point of ipsilateral stroke or any perioperative stroke or death from 11.0% to 5.1% (P = .004); however, the difference between the 2 therapies for the combined outcome of any major stroke or death was not statistically significant (25.5% for medical therapy vs 20.7% for CEA, P = .16). Major stroke was defined as persistent moderate or severe disability, vegetative state, or death. Following these results, there was an increase in CEA utilization by 30% in the year after the ACAS publication.35 However, after ACAS was published, some experts noted that the rates of mortality in ACAS were lower than those noted among nontrial participants in the same hospitals during the trial and at nontrial hospitals in the United States, prompting concerns of selection bias or lack of generalizability.36

The second of the level 1 studies in patients with asymptomatic ICA stenosis, the Medical Research Council ACST,32 which enrolled patients between 1993 and 1998, was consistent with the findings of ACAS. In ACST, 3120 patients with a 60% or more unilateral or bilateral carotid stenosis who did not have a previous CEA probable source of cardiac emboli or any major life-threatening condition were randomly allocated to immediate CEA or deferred CEA and followed up for a median of 3.4 years. Patients who were considered poor surgical risk were also excluded. Results of ACST showed an estimated reduction of the probability of any stroke or perioperative death over 5 years from 11.8% to 6.4% with immediate CEA compared with delayed CEA (P < .001). In addition, immediate CEA was associated with a significant reduction in contralateral stroke. The results of ACST were associated with increased use of CEA when compared with the years just prior to its publication.37 Multispecialty guidelines38 provide a class Ia (benefit >> risk, “it is reasonable to perform procedure”) recommendation concerning revascularization in asymptomatic patients with a stenosis of more than 70%, if the risk of perioperative stroke, MI, and death is low. However, the severity of stenosis did not predict benefit from CEA in ACAS and ACST trials (patients with a stenosis of 60% did at least as well as patients with more severe occlusive disease).31,32

The authors of the American Heart Association/American Stroke Association Primary Prevention of Ischemic Stroke Guideline used the ACST data to define absolute event rates at 5 years of follow-up, stating “Similar to ACAS, the overall rate of any stroke or death (in ACST)
was 31.2% for deferred endarterectomy vs 28.9% for immediate endarterectomy (relative risk reduction [RRR] = 7%; 95% CI, −3% to 17%; P = .172). For any major stroke or death, rates were 25.5% vs 25.3%, respectively (RRR = 7%; 95% CI, −5% to 18%; P = .242). These data must be taken into account when the procedure is considered.28

Thus, even when ACAS and ACST trials were performed, before major improvements in medical therapy, the benefits of revascularization were modest. Some subgroup findings are also worth noting. Rothwell and Goldstein39 aggregated ACAS and ACST trial data and showed that women did not benefit from CEA when compared with medical therapy. In addition, in ACST, there was no benefit from immediate CEA for patients older than 75 years. In addition, the beneficial outcomes are dependent on stringent surgical success rates of less than 3% perioperative death and stroke. This level of outcome has been hard to achieve in the general population. For example, perioperative surgical mortality in the hospitals participating in ACAS was 20% lower than in nontrial hospitals, making it unclear if the results are generalizable.36 Thus, primary care physicians should solicit the perioperative event rates of their surgeons to ensure that similar standards are met, before applying clinical trial data to their own populations.

Carotid artery stenting has had a rapid advance and integration into clinical practice during the past 2 decades.40,41 As with many new technologies, the initial decade of introduction requires the development of expertise, is marked by the rapid advance in technology, and requires a pivotal clinical trial to compare the new and old technologies. There have been 4 randomized clinical trials comparing CAS with CEA that have included patients without symptoms—the Carotid and Vertebral Artery Transluminal Angioplasty Study,42 Carotid Endarterectomy with Protection in Patients at High Risk for Endarterectomy trial,43 Carotid Revascularization Using Endarterectomy or Stenting Systems trial,44 and the Carotid Revascularization Endarterectomy vs Stenting Trial (CREST), which is a level 1 study.33 The first 3 trials used technology that is now considered out of date, had small numbers of patients without symptoms, or enrolled only a high-risk population. These 3 trials showed either no difference or a trend toward superiority for stenting.

For patients with asymptomatic ICA stenosis, CREST, sponsored by the National Institute of Neurological Disorders and Stroke and the National Institutes of Health, was the first large level 1 randomized trial in patients with standard risk to evaluate the efficacy of CAS compared with CEA.33 The study comprised 2502 patients with both symptomatic and asymptomatic carotid stenosis randomized to either CEA or CAS and included 1181 patients without any symptoms. The primary combined end point was death, any stroke, and MI during the periprocedural period and ipsilateral stroke within 4 years of follow-up (median follow-up, 2.5 years). This primary end point was similar to the main trial outcome in ACST, which included perioperative mortality, stroke, and MI and nonperioperative stroke.32 In the asymptomatic portion of the study, patients randomized to both the CAS group and CEA group had the same number of periprocedural primary end points, 21 each (Table 2).45 Moreover, there was no difference in the rate of restenosis between the 2 groups during the 2-year follow-up (6.0% for CAS and 6.3% for CEA).46 In the absence of any randomized trial evidence of superiority of either technique in patients with standard risk, the technique selection should be based on operator expertise, anatomical considerations, and patient preference. However, results of CREST have not broadened the availability of CAS. For example, Medicare does not typically cover CAS, even in situations where its use is widely

### Table 1. Level I Studies in Revascularization of Asymptomatic Carotid Artery Stenosis Disease

<table>
<thead>
<tr>
<th>Variables</th>
<th>ACAS,31 1995</th>
<th>ACST,32 2004</th>
<th>CREST,33 2010</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>1662</td>
<td>3120</td>
<td>1181</td>
</tr>
<tr>
<td>Comparison</td>
<td>CEA and OMT vs CEA alone (standard medical therapy, 325 mg/d of aspirin)</td>
<td>Immediate vs deferred CEA</td>
<td>CEA vs CEA</td>
</tr>
<tr>
<td>Inclusion criteria</td>
<td>Age 40–79 y, carotid bruit, ≥60% stenosis (unilateral or bilateral)</td>
<td>≥60% stenosis (unilateral or bilateral) that had not caused symptoms in the past 6 mo, uncertainty as to need for immediate surgery, no condition known likely to preclude long-term follow-up</td>
<td>For asymptomatic patients: ≥60% stenosis by angiography, ≥70% by ultrasoundography, or ≥80% on CTA or MRA (anatomic suitability for both techniques)</td>
</tr>
<tr>
<td>Exclusion criteria</td>
<td>Cerebrovascular symptoms, a disorder that could complicate surgery, or a condition that was likely to cause death or disability within 5 y</td>
<td>Previous ipsilateral CEA, poor surgical risk, some probable cardiac source of emboli, or any major life-threatening condition</td>
<td>Previous stroke severe enough to confound end point assessment, chronic atrial fibrillation or paroxysmal atrial fibrillation within 6 mo</td>
</tr>
<tr>
<td>Medical therapy</td>
<td>Aspirin</td>
<td>Appropriate medical care, which generally included antiplatelet therapy, antihypertensive treatment, and, increasingly in recent years, lipid-lowering therapy</td>
<td>Medical therapy that was consistent with the current standard of care, including treatment of hypertension and hyperlipidemia</td>
</tr>
<tr>
<td>Follow-up, y</td>
<td>Median 2.7</td>
<td>Mean 3.4</td>
<td>Mean 2.5</td>
</tr>
<tr>
<td>Primary end point</td>
<td>Ipsilateral stroke or any perioperative stroke and death</td>
<td>Perioperative mortality and morbidity (stroke and MI) and the incidence of nonperiprocedural stroke</td>
<td>Stroke, MI, and death during a 30-d periprocedural period or ipsilateral stroke over the follow-up period</td>
</tr>
<tr>
<td>Outcome</td>
<td>CEA vs OMT, 4.0% vs 6.2% (P = .004)</td>
<td>Immediate vs deferred, 6.4% vs 11.8% (P &lt; .001)</td>
<td>CEA vs CAS, 3.6% vs 3.5% (P = .96)</td>
</tr>
<tr>
<td>Major stroke and death</td>
<td>CEA vs OMT, 12.1% vs 13.9% (P = .16)</td>
<td>Immediate vs deferred, 25.3% vs 25.5% (P = .24)</td>
<td>CEA vs CAS, 0.3% vs 0.5% (P = .66)</td>
</tr>
</tbody>
</table>

Abbreviations: ACAS, Asymptomatic Carotid Atherosclerosis Study; ACST, Asymptomatic Carotid Surgery Trial; CAS, carotid artery stenting; CEA, carotid endarterectomy; CREST, Carotid Revascularization Endarterectomy vs Stenting Trial; CTA, computed tomography angiogram; MI, myocardial infarction; MRA, magnetic resonance angiogram; OMT, optimal medical therapy.
Please note that the text is not fully legible due to quality issues.

The presence of asymptomatic ICA stenosis indicates ischemic symptoms and biomarker elevation, biomarker elevation alone, or new Q waves alone. Cranial nerve palsy indicates injury to cranial nerves in the vicinity of the treated carotid artery that has not resolved by 6 months after the initial procedure.

agreed upon, such as an irradiated neck or a carotid artery bifurcation above C2. Currently, the availability of CAS to patients without symptoms is limited to investigational registries and clinical trials.

Within the medical community, there is a wide range of opinion concerning the value of stenting. The multispecialty guideline suggests “It is reasonable to perform CEA in asymptomatic patients who have more than 70% stenosis of the internal carotid artery if the risk of perioperative stroke, MI, and death is low, [but] it is reasonable to choose CAS over CEA when revascularization is indicated in patients with neck anatomy unfavorable for arterial surgery.”11 The multispecialty guideline also suggests that prophylactic CAS might be considered in highly selected patients with asymptomatic carotid stenosis.31 Thus, although there is controversy, revascularization may be reasonable and CAS may be appropriate in selected patients. Currently, CAS in standard risk patients is available for registry accumulation and clinical trials. However, the recently funded Carotid Revascularization Endarterectomy vs Stenting Trial 2 (CREST2) will compare optimal medical therapy with each type of revascularization.

### Current State of Atherosclerosis

Despite significant changes in natural history, medical therapies, and improvements in revascularization, the recommendations for revascularization have changed little during the past decade. Previous recommendations for revascularization suggested that a peri-procedural rate of stroke and death of less than 3% is needed to support using CEA in patients without symptoms. Current guidelines are less clear and now support revascularization if the risk of stroke, death, and MI is low.11 There is substantial controversy concerning the value of revascularization,47 because the focus lies exclusively on the low rate of stroke in these patients. However, the focus ignores changes in the natural history of atherosclerotic outcomes of these patients that have occurred synchronously with the decreasing stroke rates.

Two factors should be considered regarding revascularization of asymptomatic ICA disease: (1) the primary cause of mortality in patients with asymptomatic ICA stenosis and (2) stroke outcomes. The primary cause of death in patients with asymptomatic ICA stenosis is cardiac in origin, not stroke related. In ACST, for both groups at 10 years, there were 127 stroke deaths and 565 other vascular deaths.32 The presence of asymptomatic ICA stenosis indicates an increased risk for coronary heart disease events. Cardiac disease is the primary source of morbidity and mortality in patients with significant atherosclerosis, even when it presents in a noncardiac bed.

What accounts for these changes? One likely possibility is the improvement in medical therapy. Yeh et al48 reported that the presence of a carotid bruit increased the rate of MI by more than 3-fold to 5% per year.

The rate of stroke in asymptomatic ICA stenosis has been decreasing over the past 2 decades. This pattern of improved outcomes in atherosclerosis have been matched or exceeded by reductions in other vascular beds. Myocardial infarction in the general population has decreased by more than a third during this time.49 What accounts for these changes? One likely possibility is the improvement in medical therapy. Yeh et al48 showed that as medical therapy has improved (specifically the increased use of statins and angiotensin-converting enzyme inhibitors) in their primary prevention population, both the total number of and severity of MI has been decreasing. In addition, medical therapy that decreases MI also likely reduces stroke in asymptomatic carotid artery disease, but to a smaller absolute degree. For example, in the HPS,7 the addition of simvastatin prevented 120 coronary deaths compared with 36 non-crown vascular deaths. Similarly, in the Heart Outcomes Prevention Evaluation trial,26 ramipril reduced MI and cardiac events (heart failure, cardiac arrest, worsening angina, and coronary revascularization) more than it reduced stroke. These findings are important because the longer the expected survival, the more a patient has to benefit despite a small complication rate from a procedure. Thus, these secular changes in heart-related outcomes and cerebrovascular outcomes have altered prognosis and consideration of treatment strategies for patients with asymptomatic ICA stenosis.

The 2 major trials31,32 evaluating CEA show small reductions in stroke with CEA compared with the best medical therapy that is now out of date and the CREST trial, which showed no difference between CEA and CAS in patients without symptoms. A review of the evidence based on a Technology Assessment report commissioned by the Centers for Medicare and Medicaid Services summarizes the current state of evidence as neither sufficiently robust (for CAS) nor applicable to current clinical practice (for CEA) to determine the optimal management of patients with asymptomatic ICA stenosis.37 Thus, a new clinical trial is needed to answer the following question: Does best carotid revascularization combined with optimal medical therapy reduce death, stroke, and MI more than optimal medical therapy alone? Currently, CREST 2, a National
Institutes of Health–funded trial comparing medical therapy to each type of revascularization, will begin in the next year. CREST 2 will test medical therapy to each type of revascularization, but not to CEA and CAS directly.

In addition, the Asymptomatic Carotid Surgery Trial–2 (ACST–2) 50 comparing CAS and CEA is enrolling 5000 patients without symptoms to provide additional data comparing the 2 techniques in anatomically suitable candidates. ACST–2 will enroll patients aged 18 years or older with asymptomatic lesions deemed necessary for revascularization with a minimum 5-year lifetime expectancy whose lesion is suitable for either CEA or CAS. These data from ACST–2 may not provide the same level of information as CREST and CREST 2 because of the experience requirements of the participating physicians. In the ACST–2 protocol, vascular surgeons and interventionists both need experience from at least 25 procedures in their careers and a less than 4% stroke and death risk to qualify to participate. 50 Expertise in CAS has been shown to require experience in more than 70 cases; therefore, raising questions about the trial. 51

Follow-up of Patient
Mrs H and her physicians discussed the implications of her diagnosis of asymptomatic ICA stenosis (Table 3). Although she recognized the low rates of adverse events with any of the potential paths, she preferred revascularization therapy. She underwent uncomplicated CAS as part of a registry study, received dual antiplatelet therapy for a month, and is doing well 1 year after implantation.

### Table 3. Benefits and Risks of Each Treatment Strategy in Asymptomatic Carotid Artery Stenosis Disease

<table>
<thead>
<tr>
<th>Strategy</th>
<th>Benefits</th>
<th>Risks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal medical therapy a</td>
<td>Reductions in stroke, MI, and death</td>
<td>May not provide optimal risk reduction of stroke in all patients</td>
</tr>
<tr>
<td>Carotid endarterectomy</td>
<td>With a skilled surgeon, possible reductions in long-term risk of stroke compared with premodern optimal medical therapy alone</td>
<td>Increases risk of MI, short-term stroke risk, and cranial nerve palsy</td>
</tr>
<tr>
<td>Carotid artery stenting</td>
<td>With a skilled interventionist, similar reductions in major stroke (symptoms beyond 30 d of procedure) and lower MI rates compared with carotid endarterectomy</td>
<td>Increases rate of minor stroke (no symptoms beyond 30 d of procedure) and groin complications compared with carotid endarterectomy</td>
</tr>
</tbody>
</table>

Abbreviation: MI, myocardial infarction.

a Statin therapy, blood pressure control, tobacco cessation, and aspirin therapy are the components of optimal medical therapy.

### REFERENCES


