Stenting or Endarterectomy for Patients with Symptomatic Carotid Stenosis

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KEYWORDS

• Carotid artery stenosis • Stenting • Endarterectomy • Review
• Randomized controlled trials

KEY POINTS

- Most randomized controlled trial evidence on benefits and risks of carotid artery stenting compared with endarterectomy stems from patients with symptomatic carotid stenosis.
- In these patients, stenting causes more nondisabling periprocedural strokes than endarterectomy, but the excess stroke risk is limited to patients greater than the age of 70 years.
- Endarterectomy causes more periprocedural myocardial infarctions, cranial nerve palsies, and access site hematomas.
- Both treatments are equally effective at preventing recurrent stroke or restenosis.
- The currently available evidence does not allow making recommendations on the use of stenting for asymptomatic carotid stenosis; the results of ongoing trials comparing stenting versus endarterectomy in patients with asymptomatic disease are needed to settle this issue.

INTRODUCTION: SYMPTOMATIC CAROTID STENOSIS

In the industrialized world, stroke is the third most common cause of death, the second most common cause of dementia, and the most common reason for acquired disability in adulthood. In the Northern Manhattan Stroke Study, extracranial artery atherosclerosis was the cause of ischemic stroke in 5% of white patients, 9% of Hispanic patients, and 17% of black patients.[1] The origin of the internal carotid artery at the carotid bifurcation is the most common extracranial site of atherosclerosis

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causing stroke. Carotid disease is more frequent in men than in women and its prevalence increases with age; ultrasound screening studies in Central and Northern European and North American populations have shown a prevalence of at least a moderate degree of asymptomatic carotid stenosis narrowing the lumen by 50% or more of 2.3% in 60- to 69-year-old men, 6.0% in 70- to 79-year-old men, and up to 7.5% in 80-year-old men and older. In women, the prevalence rates were 2.0%, 3.6%, and 5.0% in the same age groups, respectively.[2] Severe carotid stenosis measuring 70% or more was present in 0.8%, 2.1%, and 3.1% in the same age groups in men, and in 0.2%, 1.0%, and 0.9% in women. Despite the high prevalence of carotid disease in the community, only a small proportion of these patients will develop transient ischemic attack (TIA) or stroke.

The pathophysiological mechanism causing cerebral ischemia in patients with carotid stenosis was long thought to be impairment of blood flow. Current understanding of the biology of atherosclerosis as well as brain imaging and transcranial ultrasound studies suggests that rupture of the atherosclerotic plaque with embolism of locally formed thrombus or plaque debris, and consecutive occlusion of distant arteries in the eye or in the brain, plays a more important role. In clinical trials, symptomatic carotid stenosis is commonly defined as having caused ischemic events in the ipsilateral eye (transient monocular blindness, so-called amaurosis fugax, or retinal infarcts) or cerebral hemisphere (TIA or stroke) in the past 6 months.

CAROTID ENDARTERECTOMY

Randomized controlled trials that enrolled patients in the 1980s and early 1990s established the benefit of carotid endarterectomy (CEA) for preventing subsequent stroke in patients with symptomatic carotid stenosis. In the *North American Symptomatic Carotid Endarterectomy Trial* (NASCET), the cumulative incidence of any ipsilateral stroke (including perioperative events) in patients with severe symptomatic stenosis (defined as 70% or more luminal narrowing) was reduced from 26% under medical therapy alone to 9% with endarterectomy, after 2 years ($P<.001$).[3] Marginal benefit was also evident in patients with moderate symptomatic stenosis (50%–69% narrowing) after 5 years, in whom surgery reduced the ipsilateral stroke risk from 22.2% to 15.7% ($P = .045$).[4] In the *European Carotid Surgery Trial* (ECST), benefit of surgery was only reported in patients with symptomatic carotid stenosis of 80% or more luminal narrowing.[5] However, this discrepancy was largely explained by differences in measurement of the degree of stenosis on angiography between the trials. In the NASCET trial, degree of stenosis was determined by comparing the most narrow vessel diameter at the site of the stenosis with the diameter of the distal normal artery.[6] In the ECST trial, the presumed diameter of the normal artery at the site of stenosis, most often the carotid bulb, was taken as the reference diameter. The method used in the NASCET trial is nowadays the most widely used method for describing the degree of stenosis.

In a pooled analysis of the endarterectomy trials, where ECST angiograms were reanalyzed using the NASCET method, endarterectomy reduced the combined outcome of periprocedural stroke or death, or ipsilateral ischemic stroke up to 5 years after treatment by an absolute difference of 15.9% in patients with severe ($\geq$70%) stenosis and 4.6% in patients with moderate (50%–69%) stenosis.[7] The risk of periprocedural stroke or death (which by definition includes outcome events occurring up to 30 days after treatment) was 7.1% in this pooled analysis and did not vary significantly with the degree of stenosis.