Introduction

Stroke is the third leading cause of death in the United States with over 783,000 strokes reported annually. Over one-third of patients die and another one-third are severely disabled. The annual economic cost exceeds $30 billion. Randomized trials have established the efficacy of carotid endarterectomy (CEA) in the prevention of stroke for patients with high-grade carotid stenosis (CS). The advent of newer technologies and a desire for less invasive treatment have encouraged investigators to propose carotid artery stenting (CAS) as an alternative to CEA. Our institution, along with others, has demonstrated that CAS is technically feasible and safe in patients with restenosis after CEA, surgically inaccessible lesions, previous radiation, or significant medical comorbidities. The 30-day stroke and death rate in 190 CAS procedures at our institution was 4.15%, indicating a competitive alternative to CEA. However, due to the proven efficacy of CEA, current indications for CAS have been limited to situations where CEA yields suboptimal results.

Two randomized trials have compared CAS and CEA. The SAPPHIRE (Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy) investigators randomized 334 high-risk patients to CAS or CEA. The 30-day composite stroke, death, and myocardial infarction rate was not different between the two groups (CAS 12.2% vs. CEA 20.1%). The European CAVATAS (Carotid and Vertebral Artery Transluminal Angioplasty Study) investigators also reported comparable 30-day combined stroke and death rates (CEA 5.9% vs. CAS 6.4%). The authors concluded that CAS was not inferior to CEA.

These trials were not powered to identify a difference between CAS and CEA. The NIH/NINDS-supported CREST (Carotid Revascularization Endarterectomy versus Stent Trial) is currently underway to make that determination. In the lead-in phase of the trial, the combined stroke and death rate was 5.6% for symptomatic and 2.4% for asymptomatic patients undergoing CAS. These preliminary results indicate low complication rates with CAS. The results of CREST may determine the role of CAS in future years. However, current data suggest clinical equipoise between CEA and CAS based on the three clinical end points of stroke, myocardial infarction (MI) and death. As a result of this information, the Food and Drug Administration (FDA) has approved the use of CAS in selected high-risk patients (significant medical comorbidities, post-CEA restenosis, anatomically inaccessible lesions above C2, and radiation-induced stenoses).

In-Stent Restenosis: The Rationale for Post-Carotid Artery Stenting Surveillance

The incidences of in-stent restenosis (ISR) after bare metal stenting of the coronary and renal arteries have been reported as 20–35% and 15–25%, respectively. It was therefore thought that ISR rates would be high with CAS too. Indeed, early reports noted post-CAS ISR in the ranges of 1–50%. However, the reported rate of ISR depends on the definition of restenosis utilized, the duration of follow-up, and the methods of diagnosis and calculation used. Most studies have relatively short follow-up periods (≤12 months), and report absolute recurrence rates weighting each procedure equally regardless of the length of follow-up. This results in an underreporting of ISR rates. With longer follow-up (1–74 months) and the use of lifetable analysis, we reported more meaningful data on ISR after CAS. The majority of restenoses ≥40% occurred within 18 months (13/22, 60%) and the majority of clinically significant restenoses ≥80% occurred within 15...
months (3/5, 60%) of their intervention (Figure 13–2). The incidence of ISR ≥ 40 and ≥60% was 42.7% and 16.4%, respectively, at 48 months of follow-up. Our data also noted that hemodynamically significant (≥80%) ISR after CAS was 6.4% at 5 years (Figure 13–3). It is clear that a significant number of patients will develop moderate ISR after CAS, of which some will progress to high-grade stenosis. There is additional evidence that the placement of a stent induces continuing arterial remodeling. Nitinol self-expanding stents can continue to expand over a 2-year period poststenting. Conversely, neointimal thickening has been reported to occur up to 1 year poststenting; further thickening may overwhelm positive remodeling of the arterial diameter from the stent and result in hemodynamically significant in-stent restenosis. This provides conclusive evidence that continued surveillance of patients is essential once CAS has been performed.

Figure 13–1. Methods of carotid revascularization practiced at our institution. (A, B) Carotid endarterectomy. Exposure and dissection of the plaque. CCA, common carotid artery; ICA, internal carotid artery; ECA, external carotid artery. (C, D) Carotid artery stenting. (B) Prestenting angiogram, arrow at stenosis in ICA; (C) poststenting angiogram, arrows at distal and proximal ends of stent.

Figure 13–2. Distribution of in-stent restenosis cases based on time of diagnosis from initial carotid artery stenting procedure. Note that the majority of restenoses occurred within 18 months of the initial carotid artery stenting procedure. The dotted line identifies the 18 month postprocedure mark. ISR, in-stent restenosis. [Adapted from Lal B K, Hobson RW 2nd, Goldstein J, et al. In-stent recurrent stenosis after carotid artery stenting: Life table analysis and clinical relevance. J Vasc Surg 2003;38(6):1162–8.]