stroke/death rate (odds ratio, 2.5; 95% confidence interval, 1.1-5.8; $P < .038$).

Conclusions: Compared with prestent ballooning alone, the use of poststent deployment ballooning increases the chances of perioperative hemodynamic instability and stroke/death rate in patients undergoing carotid artery stenting. Poststent ballooning should be limited to select populations only.

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Routine Postoperative Carotid Duplex Ultrasound Surveillance After Carotid Endarterectomy

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Introduction: Several studies have reported on the role of postoperative duplex ultrasound surveillance after carotid endarterectomy (CEA), with varying results. Most of these studies had a small sample size and/or did not analyze cost-effectiveness. Therefore, there is no consensus regarding its usefulness and whether it is cost-effective.

Methods: Analyzed were 489 of 501 CEA patients with patch closure. All patients had immediate postoperative duplex ultrasound imaging and were routinely followed up clinically and with duplex ultrasound imaging at regular intervals of 1, 6, and 12 months, and every 12 months thereafter. A Kaplan-Meier analysis was used to estimate the rate of ≥50% and ≥80% post-CEA restenosis over time and the time frame of progression from normal to ≥50% or ≥80% restenosis. The cost of post-CEA duplex surveillance was also estimated.

Results: Overall, 489 patients (mean age, 68.5 years) were analyzed. Ten of these had residual postoperative ≥50% stenosis, and 37 did not undergo a second duplex ultrasound assessment and, therefore, were not included in the final analysis. The mean follow-up was 20.4 months (range, 1-63 months), with a mean number of duplex ultrasound assessments of 3.6 (range, 1-7). Eleven of 397 patients (2.8%) with a normal immediate postoperative duplex ultrasound assessment vs four of 45 (8.9%) with mild stenosis on immediate postoperative duplex ultrasound imaging progressed to ≥50% restenosis ($P = .055$). Overall, 15 patients (3.1%) had ≥50% restenosis: nine with 50% to <80%, four with 80% to 99% (two of these had carotid artery stenting reintervention), and two had late carotid occlusion. All of these were asymptomatic, except for one who had a transient ischemic attack. The mean time to ≥50% to <80% restenosis was 14.7 months vs 19.8 months for ≥80% restenosis after the CEA. Freedom from ≥50% and ≥80% restenosis rates were 98%, 96%, 94%, 94%, 94%, and 99%, 98%, 97%, 97%, and 97% at 1, 2, 3, 4, and 5 years, respectively (Fig 1). Freedom from myocardial infarction, stroke, or death was not significantly different between patients with and without restenosis (100%, 93%, 83%, and 83% vs 94%, 91%, 86%, and 79% at 1, 2, 3, and 4 years, respectively; $P = .951$; Fig 2). The estimated cost of this surveillance was 3.6 × 489 (number of CEAs) × $800 (charge for carotid duplex ultrasound), which is $1,408,320 to detect only four patients with ≥80% to 99% restenosis who might have been potential candidates for reintervention.

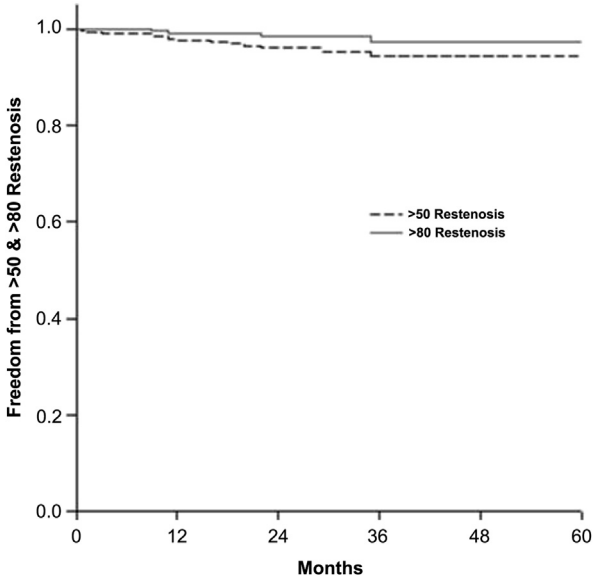

Fig 1. Freedom from >50 and >80 restenosis.

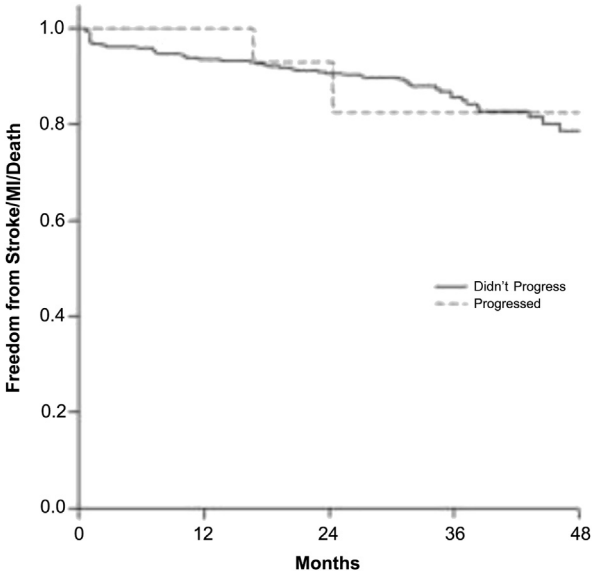

Fig 2. Freedom from stroke/myocardial infarction/death.

Conclusions: This study confirms that routine postoperative duplex ultrasound surveillance after CEA with patch closure is not necessary or cost-effective, particularly if the immediate postoperative duplex ultrasound assessment was normal or showed minimal disease.

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Risk Factors for Cranial Nerve Injury After Carotid Endarterectomy in NSQIP CEA-Targeted Database

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Introduction: Although numerous studies have described the incidence of postoperative cranial nerve injury (CNI) after carotid endarterectomy (CEA), there have been very few attempts to identify risk factors for this complication.

Methods: The 2012 CEA-targeted American College of Surgeons National Surgical Quality Improvement Program (ASC-NSQIP) database was used to determine the incidence of CNI after CEA. A patient was noted to have a postoperative CNI if his or her in-hospital or postdischarge (up to 30 days) medical record contained mention of such an injury or of symptoms consistent with such an injury. Thus, those CNIs captured by this database are likely to represent clinically relevant injuries. Multivariate logistic regression analysis was performed to identify independent predictors of CNI after CEA, using a comprehensive array of patient-, disease-, and procedure-related factors (including prior ipsilateral carotid surgery and other high-risk anatomic risk factors) as potential predictor variables.

Results: Of the 4013 patients who underwent CEA and were included in our analysis, 87 (2.2%) were noted to have sustained CNI in the first 30 postoperative days. Independent predictors of this complication included age ≥80 years (reference group <70 years; adjusted odds ratio [AOR] for CNI, 1.74; 95% confidence interval [CI], 1.00-3.03; $P = .05$), preoperative bleeding disorder, including patients in whom preoperative anticoagulation was not stopped or reversed (AOR, 1.66; 95% CI, 1.03-2.68; $P = .04$), duration of operation (AOR for each 30 minutes beyond an operative time of 90 minutes, 1.15; 95% CI, 1.06-1.25; $P = .001$), and need for reoperation (AOR, 2.65; 95% CI, 1.03-6.80; $P = .04$).

Conclusions: Our study demonstrates clinically evident CNI is a relatively uncommon event after CEA at institutions that participate in the CEA-targeted ACS-NSQIP program. Anatomic high-risk factors, including prior neck irradiation or ipsilateral carotid surgery, were not significantly associated with an increased incidence of CNI in our study, although