Eversion Carotid Endarterectomy Is Not All It Is Cracked Up to Be: It Should Not Be Widely Adopted

Thomas E. Brothers, MD, Charleston, SC

Eversion carotid endarterectomy (eCEA) has been advocated as superior to the standard operation (sCEA) with patch closure owing to technical ease, lower restenosis, and reduction in neurologic event risk. The only multicenter, prospective study to address this claim, EVEREST, randomized 1,353 patients to either eCEA or sCEA.[1] Early data suggested that restenosis of at least 50% occurred at a mean of 15 months after 2.4% of eCEA and 4.1% of sCEA (NS). At 3 years by life-table, the 5.3% of eCEA patients experiencing restenosis compared favorably with the 6.5% after sCEA. In a subsequent 4-year analysis, a significantly lower cumulative restenosis risk of 3.6% after eCEA compared with 9.2% after sCEA was announced.[2] Upon closer inspection of the data, it becomes clear that the advantage claimed by eCEA was unfairly influenced by the high rate (61%) of primary closure among sCEA patients. In fact, the 4-year restenosis rate of only 1.7% observed after patch closure with sCEA not only outperformed the 12.6% rate for primary closure but seemed to better the results of eCEA. Still, eCEA continues today to have strong proponents who would have us consider its wider application.

What are the implications for an experienced vascular surgeon considering eCEA? Because learning curves exist that represent unavoidable influences on surgical risk/benefit ratios, we are confronted with ensuring that any new technique offers some special benefit, that high standards of care are maintained; and that no patient be at increased risk. My initial personal experience with eCEA was therefore reviewed to assess its utility in my hands and to define my own learning curve. The primary outcome measures were early residual and late recurrent stenosis, with secondary outcomes of early and late ischemic neurologic events. The first 100 patients undergoing eCEA were compared with 100 contemporaneous patients using sCEA with patch closure. Operative indications were similar between eCEA and sCEA patients (63% vs 60% asymptomatic, 10% vs 7% stroke, 4% vs 5% amaurosis, 23% vs 28% transient ischemic attack [TIA]). Perioperative neurologic deficits included amaurosis (1) after eCEA, and transient cerebral ischemia (1) and retinal infarction (1) after sCEA, with 1 cardiac death each. By 3 years, one other patient in each group had suffered a TIA, but no strokes. Four carotids occluded after eCEA, compared with one occlusion after sCEA (NS). Patients undergoing eCEA showed no difference in critical (> 80%) residual or recurrent stenosis rates. However, a greater degree of recurrent > 50% stenosis was observed after eCEA at 3 years (38% vs 6%, $p < .001$) despite similar residual (early) stenosis rates. Cumulative sum analysis of recurrent > 50% stenosis showed no plateau during the first 100 eCEAs, signifying the absence of a learning curve. These findings may indicate that experience with more than 100 patients is necessary to obtain the skills necessary for optimum results with eCEA. Alternatively, they may indicate that eCEA is simply inferior in my hands. In either case, it would seem ill-advised for me to further pursue this technique. Similarly, other vascular surgeons considering eCEA would be wise to monitor their own initial results.

References

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2. Cao P, Giordano G, De Rango P, et al. Eversion versus conventional carotid endarterectomy: late results of a prospective multicenter randomized trial. J Vasc Surg 2000;31:19–30.