Can Carotid Endarterectomy Be Improved by Neurovascular Monitoring?

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Carotid endarterectomy was first proposed for stroke prevention in 1954, and in 1991 reports from the North American Symptomatic Carotid Endarterectomy Trial (NASCET) and the European Carotid Surgery Trial (ECST) showed that for specified conditions surgery was superior to medical care alone. Both trials demonstrated that despite still-remarkable perioperative morbidity and mortality rates (5.8% and 7.5%, respectively), the risk of stroke was significantly higher in patients treated with antithrombotic drugs alone for recent transient ischemic attack (TIA) and nondisabling stroke in the territory of carotid stenosis (>70% lumen narrowing).

To date, no sound scientific data exist on the challenging question of whether symptomatic patients with <70% carotid stenosis and patients without signs or symptoms of cerebral ischemia but with demonstrable obstructive lesions of the arteries supplying the brain may also benefit from carotid endarterectomy. The latter are increasingly diagnosed, and their natural history has been established by means of ultrasound follow-up studies. Annual mortality rates mainly due to coexisting coronary artery disease are high (6.6%); but depending on the rate of progression during follow-up rather than on the incidental degree of carotid stenosis, annual stroke rates are surprisingly low (progressing lesions 2.8% vs. nonprogressing lesions 1.7%). These small numbers of stroke events without preceding TIAs in asymptomatic subjects and even in symptomatic patients with <70% stenosis have resulted in the need to include more patients than might have been expected in the ongoing Asymptomatic Carotid Atherosclerosis Study Group (ACAS) as well as in the NASCET and ECST trials. For this reason as well as other problems (e.g., entry and event criteria), neither the recently published Veterans Affairs Cooperative Study Group report nor other trials on asymptomatic patients have provided conclusive data as yet.

Efficacy of carotid endarterectomy could, however, be evidenced earlier and probably more convincingly if 1) both the selection procedures of patients for surgical treatment and the definition of complications were refined beyond clinical classification alone, which could be achieved by the introduction of pathogenetically oriented ratings (e.g., determination of different stroke mechanisms such as embolism, hemodynamic compromise, and arteriolosclerosis); and 2) surgical complications were reduced by improvement of intraoperative technique and monitoring.

Surprisingly, neither issue has been addressed in the above-mentioned multicenter trials except for the general condition that participating surgeons disclosed a proven combined complication rate of less than 3%, irrespective of the details of the techniques used. Proposals to reduce the risk of thromboembolism before and after carotid clamping, to prevent intermittent perfusion ischemia, and to minimize technical failure (which represents the main cause for stroke associated with carotid endarterectomy) date back to the early 1960s: measurement of the distal internal carotid artery pressure response on common carotid artery clamping (i.e., stump pressure); local anesthesia for monitoring patient response; and electroencephalographic (EEG) recording (and more recently used, EEG mapping and evoked potential and transcranial Doppler monitoring). Although in principle these procedures seem to be of potential value to reduce the possible threats associated with carotid surgery, they may themselves introduce new risks or biases of interpretation. Advocates of intraoperative monitoring share in common the belief that by changing the surgical procedure (e.g., with the use of a temporary intraluminal shunt) stroke rates could be reduced. It has not yet been shown, however, that this concept is true although a recent retrospective, nonrandomized study offers initial support. At present, we still lack a sound scientific study addressing the issue of benefit versus risk associated with changes in surgical technology as a result of intermittent neurormonitoring. This continuing dilemma is again raised by the report published in this issue of Stroke by Jansen et al. The authors report their conclusions from 130 carotid endarterectomies (<65% stenosis, n=15; >65% stenosis, n=115). Unfortunately, inherent shortcomings in the design of this study limit the validity of its results: it includes patients with different types of cerebrovascular ischemia (amaurosis fugax, TIA, and nondisabling stroke [63%] as well as asymptomatic subjects [37%]) despite an established fivefold to tenfold higher surgical morbidity in patients with preoperative stroke. The small number of patients, which results in an even smaller number of ischemic events, impedes any statistical data interpretation. Assuming that EEG asymmetries of a certain degree indicate the need to insert a shunt, only seven patients indicated hemodynamic changes on TCD monitoring alone; nine patients had similar changes but were shunted because of simultaneous EEG changes. Two patients remained free of events.

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in spite of a complete disappearance of Doppler signals, whereas one patient with only reduced flow velocities developed a stroke. Even less convincing are the TCD findings with respect to the capacity of TCD to display cerebral embolisms, which the authors believe are warning signs that justify modification of the surgical procedure: 75 intermittent, high-amplitude Doppler signals recorded in 55 patients before and after endarterectomy remained fortunately without deficit, and one patient suffered a mild reversible neurological deficit without infarction confirmed by computed tomography.

How can TCD monitoring potentially make carotid endarterectomy safer, and what are its present limitations? For the assessment of hemodynamic problems, this information may extend preoperative TCD studies with short compression tests of the common carotid artery on intraoperative conditions with longer arterial blocking, anesthesia, and technical problems (e.g., carotid thrombosis). This may be used to survey adequate vascular perfusion before the completion of surgery if immediate procedures such as surgical revascularization or medical thrombolysis could be implemented. However, measuring flow velocity in a limited segment of the intracranial carotid territory fails to provide conclusive insight into total tissue perfusion, and correlations between findings and any clinical event should be cautiously made to avoid misinterpretation. Similarly, the interpretation of high-intensity Doppler signals as hazardous cerebral embolisms may be rather premature; in cardiovascular surgery for years numerous such events have been registered without significant clinical deterioration. The same has recently been shown during early (2–6 hours) spontaneous lysis of intra-arterial clots in acute stroke and during intra-arterial injections. Whether different constituents of such signals might have different clinical relevance is an open question.

What can be done to improve this technique in the future? Additional analysis of Doppler signal intensity rather than flow velocity alone may allow blood flow volume and vascular anatomy calculations, which probably better correlate with cerebral perfusion. Recordings from plane sections using duplex scans with multiple rather than single stationary sample volume analysis may extend the scope to cerebral arteries adjacent to the circle of Willis (anterior and posterior cerebral arteries) and allow proximal and distal arterial recordings, with particular attention to branching segments supplying the deep vascular territories. Whether the information provided can guide the surgeon to lower complication rates remains to be carefully investigated.

**References**

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