Regarding “High embolic rate early after carotid endarterectomy is associated with early cerebrovascular complications, especially in women”

A. Ross Naylor, MD, FRCS, Leicester, United Kingdom

Postoperative carotid thrombosis (POCT) complicates 2% to 3% of carotid endarterectomies (CEAs) and classically occurs 4 to 6 hours after restoration of flow. It was previously held that POCT was almost invariably associated with some underlying technical error and incurred a poor outcome unless thrombectomy was performed within 1 hour. Viewed traditionally as being unpreventable, it would clearly be preferable to identify patients at high risk early and so intervene (therapeutically or surgically) to prevent progression onto thrombosis and stroke. The paper by Laman et al, in this edition of the Journal, is now the latest of five around the world1-6 to suggest that many of the traditionally held views regarding POCT need revising and that transcranial Doppler (TCD)–directed embolus monitoring may be invaluable in identifying those at highest risk.

The available evidence suggests that a policy of quality control assessment will virtually abolish intraoperative stroke (apparent on recovery from anesthesia) but that this will have surprisingly little effect on the rate of stroke as the result of POCT.7 Secondly, provided technical error has been excluded with some form of completion assessment (eg, angioscopy), patients who subsequently have a stroke because of POCT are unlikely to have underlying technical error at reexploration.3 Interestingly, most are found to have platelet-rich thrombus that is adherent to the endarterectomy zone rather than to the patch. It is now well accepted that platelets begin to adhere to the endarterectomy zone within minutes of flow restoration,8 but no one knows why it becomes excessive in certain patients. Thirdly, there are now five studies (including that of Laman et al) that suggest patients destined to progress to stroke because of POCT have a 1-hour to 2-hour period of increasing embolization before any neurologic deficit becomes apparent.1-6 The prevailing view is that, as the platelet thrombus accumulates, small particles are increasingly shed into the carotid circulation and detected with the TCD. Overall, about 50% of patients with CEA will have one or more emboli detected in the postoperative period, but only about 5% will progress to high-grade sustained embolization.7 In Laman et al’s series, 30% of these patients with high embolization progressed to stroke, increasing to 50% in the Leicester series3 and 60% in Levi et al’s4 Australian study.

Unfortunately, because of methodologic differences among the five studies (varying monitoring times [10 to 180 minutes], divergent antiplatelet and reexploration policies [especially in Laman et al’s study] and differing policies regarding intervention [Boston gave dextran to all patients with CEA, Leicester administered dextran 40 to all patients with high-rate embolization]), it is currently impossible to combine the data and present an overview of the principal findings. This problem may, however, be resolved in the future when a collaboration of interested researchers reanalyze all of the raw data from the various major studies. Laman et al’s study proposes that an embolus rate of 0.9/min represents a threshold for increased risk, and in Leicester, we would only intervene if the embolus count exceeded 25 in any 10-minute monitoring period (2.5 emboli/min) or where the embolus distorted the waveform suggesting that it was large.7 Our threshold of 25 in 10 minutes was based on Gaunt et al’s original work.8 To date, we have not observed any early embolic stroke in anyone with an embolus rate of between 0.9/min and 2.5/min, which is clearly at variance with Laman et al’s findings, although part of the discrepancy might be explained by our use of completion angioscopy and the substitution of aspirin with warfarin 1 week before surgery in the Dutch center.

However, despite the methodologic problems, these five studies should now be the catalyst for studying the phenomenon of POCT in a more standardized manner, especially regarding antiplatelet regimes (see below).
particular, we do need to know: 1, whether other centers have unpublished data on postoperative monitoring that will further our knowledge; 2, whether certain people appear to be at increased risk of POCT; and 3, what the best treatment is for patients with high-grade embolization.

Contrary to traditional teaching, current research now suggests that it is the patient who is probably prothrombotic. Patients at high risk might therefore be identified before surgery in the future. Evidence supporting this hypothesis includes the following observations: 1, postoperative embolization is unrelated to patch type (prosthetic versus vein); 2, patients undergoing staged bilateral CEAs have similar magnitudes of embolization after each procedure (if the patient had a high embolization after the first CEA, the same is likely after the second); and 3, the magnitude of embolization is unrelated to aspirin therapy, but the platelets of patients with high rate embolization are significantly more sensitive to adenosine diphosphate stimulation. In addition, Laman et al’s observation that female patients have a higher rate of postoperative embolization (than male patients) is important and could explain why women traditionally have a higher operative stroke rate than men.

There is also no current consensus as to the best way of treating patients at high risk for POCT. A number of options are available including: 1, routine intravenous dextran 40 therapy; 2, selective dextran 40 therapy to patients with high-rate embolization; 3, intravenous S-nitroso glutathione; 4, intravenous rheopro; or 5, intravenous heparin (as was used in Laman et al’s study).

The disadvantage of routine dextran is that up to 36% of patients with high-rate embolization need an increase in the dose to control the excessive rates of embolization. Despite administering routine dextran to their patients with CEA after restoration of flow, up to 20% of the Boston patients still had one or more emboli detected after surgery and POCT was still observed. Of more practical importance, dextran should not be administered to patients with significant chronic renal impairment because of risk of precipitating multiorgan failure. We have observed a major change in surgical practice since we introduced a program of 3-hour TCD monitoring and selective dextran 40 therapy in October 1995. Since then, dextran has only been administered to 4% of patients and 850 CEAs have been performed. No one during this time has had a stroke as a result of POCT. On the basis of previous experience, we might otherwise have expected to have reexplored 17 to 25 patients for postoperative thrombotic stroke.

In conclusion, despite many false dawns, TCD appears at last to have found a valuable role in the prediction and prevention of POCT. Although some centers may wish to use TCD to identify patients at high risk in the early postoperative period, it seems likely that the optimal role for TCD in the future will be to enable clinicians to develop novel antiplatelet strategies (preferably administered before surgery) so that, ultimately, no postoperative monitoring is necessary at all.

REFERENCES


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Please see related article by Dr D. Martin Laman et al on pages 278-84.