Commentary

The Challenge of Carotid Occlusion

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The authors of the preceding reports [1–3] are to be commended for their efforts to solve a problem that has plagued medicine for almost 100 years, that is, the need to know if it would be safe to sacrifice the carotid artery. Struggling with this problem has been frustrating. The experience of having a patient undergo successful carotid test occlusion and subsequent permanent occlusion, only to have a stroke a few hours later is quite disheartening. Anyone who has performed a number of carotid occlusions knows that even though this is a relatively simple procedure, the complication rate is alarmingly high.

It is thought that infarction after successful temporary and then permanent carotid occlusion has two basic causes. One is hypoperfusion and ischemia due to inadequate collaterals. The other major cause is embolism, due to clot developing in the stump of the occluded supraclinoid carotid artery and then dislodging and entering the middle cerebral artery circulation. The preceding papers address the problem of hypoperfusion; this is affected by patency of the circle of Willis and by the stability of blood pressure and cardiac output.

Studies with xenon CT made a noble effort to address this problem in the past, but the results were never conclusive. Also, the difficulty of performing xenon CT greatly limited the wider application of this technique. With the development of HMPAO-SPECT we now have a tool that is widely available, and this gives us an opportunity to answer the question originally raised by xenon CT.

With injection of HMPAO during temporary carotid occlusion, it is hoped that asymmetry of perfusion to the hemispheres will be more valuable than temporary carotid test occlusion alone in predicting which patients will tolerate permanent carotid occlusion. The present delayed stroke rate after temporary test occlusion that leads to permanent test occlusion is unacceptably high, in the range of 10–20%. Two main factors affect the delayed hypoperfusion infarct that we hope can be predicted on the basis of the results of HMPAO SPECT studies. One is the adequacy of the collaterals around the circle of Willis; the other is the cardiovascular status of the patient. This problem of hypoperfusion is definitely more serious in the elderly, whose blood pressure and cardiac output are less stable than in younger persons.

The other major cause of delayed stroke after a normal carotid test occlusion is emboli from the supraclinoid carotid stump. It is not known if injection of heparin after carotid test occlusion will reduce the prevalence of emboli. In the past, most carotid occlusions were done surgically, and physicians were reluctant to give heparin to postoperative patients. With balloon occlusion of the carotid artery, use of heparin after occlusion is not a problem. Controlled studies are needed to determine if anticoagulation will decrease the number of emboli. Another recent development may help in the detection of emboli. Transcranial Doppler sonography can be used now to detect emboli in the major intracranial vessels. This obviously will serve as an adjunct in the management of patients after carotid occlusion. Studies are currently under way to determine what percentage of patients have intracranial emboli after carotid occlusion. Another factor that affects the propagation of emboli is balloon placement distal relative to the origin of the ophthalmic artery. In a number of patients, reversal of flow in the ophthalmic artery with restoration of antegrade flow in the supraclinoid carotid occurs after carotid occlusion. This antegrade flow probably does dislodge emboli

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from the clot that is forming in the supraclinoid carotid artery. A large number of patients can tolerate occlusion of the ophthalmic artery without blindness developing, but, again, no definitive study has been done to access the reliability of temporary ophthalmic test occlusion as an indicator of whether blindness will result.

Obviously a number of variables need to be studied to find out what can be done to reduce the prevalence of complications associated with occlusion of the carotid artery. Functional reserve can be studied by using HMPAO. With the development of transcranial Doppler sonography, the role of emboli in carotid occlusion can be studied more effectively. Factors such as cardiac output and ejection fraction and their relationship to the stability of blood pressure may also have high predictive value. Finally, the simple factor of age may eventually turn out to be the best predictor of all.

Any study of carotid occlusion probably should be done with balloon occlusion. Ligation of the carotid artery usually involves complicated surgery, and therefore many other variables are added that could increase the complication rate.

Another more difficult question is, Which is better for the patient, preoperative or intraoperative occlusion of the carotid artery? Often the surgeons are not certain that they will need to sacrifice the carotid artery at surgery. The question arises, Is it safer to occlude the artery preoperatively or intraoperatively? The first probably results in more carotid occlusions overall. The second may result in overall fewer occlusions but probably also results in a higher delayed stroke rate because of the inability to give patients heparin postoperatively and because of the alterations in blood pressure associated with general anesthesia.

As humans were meant to have internal carotid arteries to nourish the most important organ in the body, occlusion of these vessels will never be easy. Only through functional studies such as those reported in the preceding papers will we be able to make carotid occlusion a safer procedure.

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