Risks and Benefits of Shunting in Carotid Endarterectomy

James H. Halsey Jr., MD,
for the International Transcranial Doppler Collaborators

Background and Purpose: Controversy continues about the pathogenesis of perioperative stroke in carotid endarterectomy and the use of shunting. The purpose of this study was to determine, using transcranial Doppler ultrasonography, the severity of ischemia during clamping of the carotid artery as a basis for analysis of complications in patients operated on with and without shunting.

Methods: In a retrospective study, 11 centers contributed 1,495 carotid endarterectomies monitored with transcranial Doppler. The cases were divided into groups with severe, mild, and no ischemia, and each group was subdivided according to shunt use. The perioperative rate of severe stroke attributable to intraoperative ischemia, in addition to total perioperative stroke, was determined for each subgroup.

Results: Severe ischemia occurred in 7.2% of our cases but cleared spontaneously in about half of these. In those with persisting ischemia the rate of severe stroke was very high, while shunting protected against stroke in such cases. If ischemia did not occur, the stroke rate was higher with shunting, although not so high as in unshunted cases with severe ischemia. Slightly more than one third of the severe strokes were due to postoperative cerebral hemorrhage or carotid thrombosis, unrelated to clamp-induced ischemia or shunting.

Conclusions: Carotid endarterectomy complications might be reduced by selectively shunting only for severe persisting ischemia. Monitoring of cerebral ischemia would be essential to selective shunting. (Stroke 1992;23:1583-1587)

KEY WORDS • carotid endarterectomy • cerebral ischemia • ultrasonics

Carotid endarterectomy has been shown to be superior to medical management of symptomatic severe carotid stenosis if surgical morbidity and mortality are low.1-3 However, controversy persists about the pathogenesis of surgical complications and the proper use of shunting to reduce them: whether to shunt always to prevent ischemic damage,4 to shunt never because significant ischemia occurs too infrequently to be worth the technical hazards of shunting,5,6 or to shunt selectively based on hemodynamic or electrophysiological monitoring for detection of the minority of cases in which serious cerebral ischemia occurs, believing that in these cases the risk of infarction is high if the ischemia is prolonged.7

It has not been possible to resolve this controversy because the complication rate of carotid endarterectomy is now so low that no surgical group can collect sufficient cases to demonstrate a conclusive advantage of any one policy. A critical factor confounding the debate has been the difficulty of measuring cerebral ischemia as the essential element upon which the assessment of shunting should be based.

Sundt,7 who pioneered selective shunting based on regional cerebral blood flow (rCBF) and electroencephalographic (EEG) monitoring, achieved very low morbidity and mortality. He argued that failure to shunt in approximately 30% of cases would have been associated with a high probability of infarction.7 However, his large series contained few nonshunted severely ischemic cases and no shunted nons ischemic cases.

To address this controversy, we have retrospectively analyzed data from several centers engaged in transcranial Doppler (TCD) monitoring. No center had sufficient cases to yield any conclusion beyond the technical feasibility of monitoring and the conviction that monitoring helped classify the pathogenesis of complications.8-11 Combining the results of all centers yielded a large number of cases. Because there was a diversity of shunting practices while all cases had TCD monitoring, it was possible for the first time to analyze the impact of shunting in the presence of different degrees of cerebral ischemia.

Subjects and Methods

Eleven centers (see "Appendix") contributed 1,495 carotid endarterectomies in which TCD monitoring was performed. Indications for surgery varied among centers but generally included carotid stenosis comprising >50% reduction of cross-sectional area, usually with history of transient ischemic attack or minor stroke, although asymptomatic patients were included at some centers. Patients with contralateral occlusion were excluded at some centers, as were virtually all cases of recent and severe stroke at all centers. Anesthesia was general endotracheal with isoflurane, enflurane, or halothane (sometimes supplemented by nitrous oxide).
in the majority of cases. In one center the majority, and in another center about half, together comprising 125 cases, were operated on under local anesthesia. Seventy-five cases, mostly from one center, were performed under very deep anesthesia, to cause an EEG suppression–burst pattern.

The TCD monitoring was performed with either an EME TC 2-64 (Uberlingen, FRG) or a Medasonics Transpred (Freemont, Calif.) device. Patients were not included if an interpretable velocity waveform could not be obtained, as was the case in 5–15% of patients attempted at different centers. This is a function of the patient’s age, sex, and race and the sonographer’s skill.12

The recording consisted of the velocity waveform at a depth from the temporal scalp of 55–35 mm, representing the middle cerebral artery (MCA) or one of its branches. Recordings deeper than 55 mm were likely from the carotid siphon. These were excluded since at clamping the velocity could fall to zero, even though collateral perfusion of the MCA remained adequate from the posterior communicating artery or the anterior cerebral artery. Recordings more superficial than 45 mm were likely from a branch of the MCA, but these were a satisfactory index of MCA perfusion when the velocity after clamping was expressed as percent of that before clamping.11 Patients were also excluded if it was thought that the posterior cerebral artery had been insonated. The main criterion for this, in addition to angle of insonation, was the observation that mean velocity (MV) increased at carotid clamping, representing collateral blood flow, without a blood pressure increase sufficient to account for it. MV was calculated as DV+ (SV–DV)/3, where SV and DV denote the peak systolic and end-diastolic velocities, respectively.

In all cases MV was recorded just prior to clamping and within the first minute after clamping and in some cases at 5 minutes after clamping. In shunted cases, an effort was made to record the time from clamping to shunting, MV during clamping prior to shunting, and the responses to shunting and the shunt’s subsequent removal.

Ischemia was called severe if MV during the first minute after clamping was 0–15% of the preclamp value, mild if 16–40%, and absent if >40%. These ranges were chosen early in the study, influenced by ongoing correlation recordings of rCBF, EEG, and carotid stump pressure being made at some of the centers.8–11

Use of shunting varied among surgeons, even within a center, according to the judgment of the surgeon. Some did not shunt in any case, some did in all cases, and the majority of surgeons were selective in shunting, based on varying criteria including the severity of ischemia, EEG change or carotid stump pressure if these were being recorded, loss of consciousness in the local anesthesia cases, and anticipated duration of the clamping. Some surgeons gradually modified their practices. For example, some surgeons who had always shunted began to omit this if TCD indicated no or only mild ischemia, while some who never shunted began to do so when severe ischemia occurred. Successful shunting was defined as placement of the shunt, resulting in MV of >40%, within 5 minutes and restoration of blood flow within 5 minutes after shunt removal. If attempted shunting was unsuccessful, the case was counted as not shunted.

Strokes recognized within the first week after surgery were classified as mild if the disability cleared completely within 24 hours or was so slight as to not interfere with the patient’s self-care and independence in dressing, bathing, feeding, or toilet. Strokes were counted as severe if persisting disability required the patient to have assistance in performing one or more of these functions. For the purpose of this study, cranial or peripheral nerve palsies and wound hematomas requiring surgical drainage were not counted. In most centers a neurologist was not involved in preoperative and postoperative evaluation, although most of the recognized strokes were evaluated by a neurologist. To minimize variability in disability recording, the primary analysis has been based upon occurrence of severe stroke, a robust observation resistant to bias. The TCD measurement was quantitative and objective, also resistant to bias. Testing of statistical significance was by calculation of $\chi^2$.

### Results

Among 1,495 carotid endarterectomies, 81 perioperative strokes occurred (5.4%); 44 (2.9%) were mild and 37 (2.5%) were severe, five being fatal. Of the severe strokes, five were due to intracerebral hemorrhage, nine to carotid artery occlusion, and 23 to intraoperative ischemia, either hypoperfusion during clamping or embolism (Table 1). In addition, two deaths due to myocardial infarction and one due to heart failure occurred. Reporting of nonfatal myocardial infarction was incomplete. Virtually all strokes comprised hemiparesis or hemiplegia contralateral to the side of surgery. Exceptional cases included two upper brain stem infarcts (one mild and one severe), both in patients with contralateral carotid artery occlusion. In these patients it was thought that the brain stem was at the watershed area between the carotid and basilar systems. One case of severe hemiparesis occurred ipsilateral to the side of surgery in the presence of contralateral carotid artery occlusion. Computed tomography revealed a watershed infarct on the side of the occlusion. Severe bilateral EEG suppression occurred in all three of these cases, associated with MV of <15%. Four carotid artery occlusions were recognized during surgery, manifested by falling MV late in the procedure or failure of MV to recover on clamp release. Three of these occlusions were intimal flaps that were repaired and one was a carotid thrombus distal to the clamp, removed with a Fogarty catheter. Since these occlusions were corrected during surgery and did not cause disability, they were not counted as complications.

### Table 1. Perioperative Stroke Among 1,495 Carotid Endarterectomies

<table>
<thead>
<tr>
<th>Complications</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>44</td>
<td>2.9</td>
</tr>
<tr>
<td>Severe</td>
<td>37</td>
<td>2.5</td>
</tr>
<tr>
<td>Postoperative hemorrhage</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>Postoperative carotid occlusion</td>
<td>23</td>
<td></td>
</tr>
<tr>
<td>Intraoperative ischemia</td>
<td>37</td>
<td>2.5</td>
</tr>
<tr>
<td>Total</td>
<td>81</td>
<td>5.4</td>
</tr>
</tbody>
</table>

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The 1,495 patients were grouped according to degree of ischemia at clamping (Table 2). Among the centers the incidence of severe ischemia varied from 0% to 21%. This variability was partly a function of the frequency of contralateral carotid artery occlusion (Table 3), in which situation the incidence of severe ischemia was 26%.

Severe ischemia occurred in 107 cases, MV immediately falling to <15% at clamping. In some cases, MV then began to rise spontaneously, reflecting opening of the collateral circulation. In other cases, not shunted, blood pressure was elevated, sometimes resulting in a rise in MV, converting some cases of initially severe ischemia to mild or no ischemia within a few to several minutes. At clamp release there was brisk recovery, usually with transient hyperemia. Successful placement of a shunt also caused brisk recovery of MV, always to >40% and usually to >80%.

Among the severe strokes in unshunted severely ischemic cases, one was attributed to embolism occurring a few seconds prior to clamp application, associated with ipsilateral EEG suppression. Brisk back-bleeding was observed after clamp application, although MV remained zero. Reperfusion did not occur on clamp release but developed gradually, beginning about 10 minutes later, followed by gradual EEG recovery. The five cases of severe stroke attributed to hypoperfusion included two in which shunting was attempted but not established within 5 minutes, with effective clamp times of 5 and 11 minutes. For the other severe strokes occurring in unshunted severely ischemic cases, clamp times were 8, 13, and 28 minutes. In 15 severely ischemic cases mean±SD clamp time was 21±9 minutes, with no difference between those with and those without complications. For 38 initially mildly ischemic cases mean±SD clamp time was 23±15 minutes and for 420 nonischemic cases 26±15 minutes.

Although clamp time was not related to complications, in initially severely ischemic cases the rate of

| Table 3. Ischemia, Contralateral Carotid Artery Occlusion, and Shunting Among Centers |
|-------------------------------|---------------|-----------------|-----------------|---------------|---------------|-----------------|---------------|
| Shunting                     | Stroke        | Shunting        | Stroke          | Shunting      | Stroke        | Shunting        | Stroke        |
| Total cases (No.)            | Severe        | Contralateral   | Shunts          | No.           | %             | No.             | %             |
|                              | ischemia (%)  | occlusion (%)   | %               | No.           | %             | No.             | %             |
| 1                            | 342           | 3.5            | 13              | 5             | 1.5           | 1               | 20.0          |
| 2                            | 298           | 8.7            | 17              | 46            | 15.4          | 2               | 4.3           |
| 3                            | 247           | 9.7            | 17              | 148           | 59.9          | 4               | 2.7           |
| 4                            | 175           | 11.4           | 10              | 18            | 10.3          | 0               | 0.0           |
| 5                            | 95            | 0.0            | 0               | 12            | 12.6          | 1               | 8.3           |
| 6                            | 85            | 21.2           | 15              | 21            | 24.7          | 0               | 0.0           |
| 7                            | 82            | 1.2            | 11              | 13            | 15.9          | 1               | 7.7           |
| 8                            | 76            | 2.6            | *               | 9             | 11.8          | 0               | 0.0           |
| 9                            | 62            | 3.2            | 0               | 14            | 22.6          | 0               | 0.0           |
| 10                           | 23            | 0              | *               | 0             | 0             | 0               | 0.0           |
| 11                           | 10            | 20.0           | 20              | 1             | 10.0          | 0               | 0.0           |

*Arteriographic data not available.
recovery from ischemia was critical. An index of this important variable was retrieved in 26 cases. There were 13 cases with an initial MV of <15% in which MV did not recover to >40% by 5 minutes after clamping; all five severe strokes attributed to hypoperfusion, in addition to the case of MCA embolism (which occurred just prior to clamping), occurred among these cases. There were no strokes among the 13 cases in which the severe ischemia had resolved to >40% within 5 minutes (p<0.01, Table 2).

The one unshunted, initially mildly ischemic patient who suffered a severe stroke had both initial and 5-minute MVs of 24% and a clamp time of 35 minutes. This case was complicated by the presence, preoperatively, of bilateral anterior cerebral artery occlusions. Severe ipsilateral EEG suppression occurred during clamping. Five other initially mildly ischemic patients tolerated a MV of <40% at 5 minutes without complication.

Use of shunting varied widely, among the largest centers from 1.5% to 59.9%, only distantly related to the frequency of severe ischemia (Table 3). Shunting conferred protection against severe stroke due to severe ischemia, which occurred initially in 107 cases (7.2%, Table 2). There were no severe strokes among 74 cases of severe ischemia at clamping if a functioning shunt was established within 5 minutes of clamping, compared with six strokes among 13 cases with persisting ischemia that was initially severe (46%, p<0.0001). From this small subgroup it is clear that severe ischemia, if not rapidly resolved spontaneously by induced hypertension or by shunting, is very serious.

Although it happened that no strokes occurred among the shunted severely ischemic cases, shunting nonetheless was not a benign procedure. There were nine severe strokes among the 287 shunted cases (3.1%). Because of the few mildly ischemic cases, the difference between shunted and unshunted was not significant. But among nonischemic cases, six of 136 shunted suffered severe stroke (4.4%) while seven of 1,016 unshunted (only 0.7%) did so (p<0.001, Table 2). The difference in stroke rates between severely ischemic shunted and nonischemic shunted cases was not significant (0.10>p>0.05).

Including mild strokes, the severely ischemic group comprised eight total strokes among 74 shunted cases (10.8%) but 11 among 33 unshunted cases (33%). This difference was significant (p<0.05) but less strong than for only severe strokes. Data could not be retrieved to permit analysis of a separate subgroup of mild strokes resulting from persisting initially severe ischemia. For cases with mild ischemia the total stroke rate was virtually the same with or without shunting (six of 77 and 10 of 159, respectively). For nonischemic cases the effect of shunting was unfavorable, with 12 total strokes among 136 shunted cases (8.8%) but 21 among 1,016 unshunted (2.1%, p<0.001).

There was no relation between shunting and postoperative carotid thrombosis or intracerebral hemorrhage. Complications were too few to analyze the effects of anesthesia on outcome.

Discussion

These data show that in experienced hands severe ischemia could be demonstrated by TCD monitoring. Within the limitations of retrospective review, it appears that most patients have adequate collateral circulation to tolerate up to at least 30 minutes of unilateral carotid clamping without suffering ischemic damage. In these, shunting is unnecessary and the small risk of stroke due to it makes it undesirable. However, in the few patients in whom severe ischemia occurs and persists, the small risk of shunting is justifiable to protect against the very large risk of ischemic infarction. This tentative conclusion might form the basis for confirmation and amplification in a subsequent prospective study.

An important limitation in this study is that the disability was recorded mostly by the vascular surgeons caring for the patients, without involvement of a blinded neurologist. Realizing the risk of underreporting of minor neurological disability, we chose severe stroke (severe hemiparesis or hemiplegia in virtually all cases) as the dependent variable. This is a robust observation, detectable even by laymen, including patients themselves, and by nurses. It should be resistant to error. In fact, it revealed the largest differences, while differences involving mild strokes were less significant. The morbidity considered was only the perioperative complications. While this would be less desirable than 30-day morbidity in a comparison of surgical and medical management, for the hemodynamic question addressed perioperative complications may be preferable. Any late morbidity attributable to shunting should strengthen the impression of its undesirable effects while the protective role of shunting should be strongest against strokes evident at the end of the procedure.

A second limitation is that shunting was not randomized. This certainly made the total stroke rate sensitive to selection criteria for surgery and the use of shunting, specifically including the influence of TCD monitoring on the decision to shunt. Indeed, as the study progressed monitoring caused a "drift" toward more shunts in severe ischemia and fewer in the absence of ischemia, with more cases with contralateral carotid artery occlusion being accepted for surgery at some centers. This reduced the number of unshunted cases with severe, persisting ischemia and of shunted cases with no ischemia and hence of total complications that would have occurred in a randomized study. However, the total stroke rate comparing shunted and unshunted cases was not the dependent variable. The dependent variable was, instead, the separate stroke rates within each subgroup defined by degree of ischemia. It is a fundamental hypothesis of this study that randomization would affect subgroup sizes but not the stroke rates within subgroups, i.e., that risk factors determining stroke operate primarily by determining severity of ischemia. If a randomized trial of shunting were to be attempted to test this hypothesis, it is clear from our study that randomization should be stratified according to the severity of measured ischemia.

The appearance of disparity in severe stroke rates between shunted severely ischemic (0 of 74) and shunted nonischemic (six of 136, 4.4%) cases might imply nonrepresentative weighting of one or another of the subgroups. The difference between these subgroups, however, was not significant (0.10>p>0.05). There is no reason that shunting should be less dangerous in severe ischemia. Indeed, if mild strokes were included, the total stroke rate for shunted severe ischemia (eight of 74, 10.8%) was virtually the same as for shunted no ischemia (12 of 136, 8.8%). If some strokes had oc-
ecurred in severely ischemic shunted cases, suppose three of 74 (4.1%), the benefits of shunting versus not shunting all cases of initially severe ischemia would still be significant \((p<0.05)\) while protection against persisting ischemia (five of 12, 42%, even excluding the embolic case) would remain very strong \((p<0.001)\).

The first important conclusion from this study is that severe persisting ischemia is infrequent but very serious when it does occur. Presuming accurate hemodynamic measurements, shunting is clearly needed to protect against it. This condition is so dangerous that a shunt complication rate of 10–20\% would still demonstrate benefit, while in the absence of ischemia a complication rate of 2\% would not be acceptable from a conceptual point of view and could be justified in practice only if monitoring were unavailable.

The second important conclusion is that shunting is an important stroke risk factor. The range of shunt complications among our centers suggests that these can be reduced with practice. Although the rate was 0\% at the five centers, these included only 63 total shunts, a rate in fact not significantly different from the 2.7\% rate among the 148 shunts at center 3 \((p>0.10)\).

An important gap in our data should be noted. Although shunting was not beneficial in mild ischemia (MV of <40\%), this should be qualified by the short clamp times in the majority not shunted. A protective role for shunting in mild ischemia with clamp time approaching an hour, presuming persistence of mild ischemia, remains possible. This consideration might account for the report of a lower complication rate in 63 shunted patients compared with 75 not shunted.\(^4\) In these cases, cared for in the context of a residency training program, clamp times might have been longer than in ours. More data will be required to clarify this.

Quantifying the severity of ischemia clarifies the pathogenesis of perioperative stroke. The strong association of unshunted, persisting severe ischemia with stroke argues for a hemodynamic pathogenesis in this subgroup. The simultaneous occurrence of EEG flattening and of hemiplegia or coma in local anesthesia cases supports this argument. Conversely, it seems reasonable to invoke an embolic mechanism in those other strokes not associated with ischemia. We presume that strokes attributable to shunting are embolic in pathogenesis, most likely due to dislodgment of atheromatous and thrombotic material by the shunt tubing. Without proof, this hypothesis is at least consistent with the much lower frequency of stroke in nonischemic unshunted cases. Two other groups of complications, postoperative hemorrhage and postoperative (and intraoperative) carotid artery occlusion, are also identifiable, not related to clamp ischemia or shunting. They should be separately analyzed to elucidate pathogenesis and prevention.

Appendix

Contributing investigators (in alphabetical order): R.G.A. Ackerstaff, St. Antonius Hospital, Koekoekslaan, The Netherlands; Malcolm P. Berger, Neurology Associates, Union and Fine Avenues, Brackenridge, Pa.; Michael H.F. Edelmann, Department of Surgery, Knappschaftskrankenhaus, Ruhr-Universitat Bochum, Bochum, FRG; Donald M. Fisher, 570 Ridge Road, Lackawanna, N.Y.; James H. Halsey, Department of Neurology, University of Alabama Medical Center, Birmingham, Ala.; John W. Norris, Sunnybrook Medical Centre, Toronto, Canada; Shirley Otis, Department of Neurology, Scripps Clinic, La Jolla, Calif.; John A.H. Porter, 2002 West Howard Avenue, Milwaukee, Wis.; E. Bernd Ringelstein, Neurologische Klinik, Technical University Aachen, Aachen, FRG; Torben Schroeder and Lisbeth Jorgensen, Department of Vascular Surgery, Rigshospitallet, Copenhagen, Denmark; Merrill P. Spencer, Institute of Applied Physiology and Medicine, Seattle, Wash.; and H.J. Steiger, Neurochirurgische Klinik, Inselspital, Berne, Switzerland. Drs. Edelmann and Ringelstein collaborated as a single investigative team.

References


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J H Halsey, Jr

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