Operative Mortality Following Carotid Endarterectomy

To the Editor:

I read with great interest the editorial by Dr. Gary G. Ferguson in the May-June 1982 issue of STROKE, Volume 13, p. 287, entitled "Intraoperative Monitoring and Internal Shunts: Are They Necessary in Carotid Endarterectomy?". I was dismayed, however, by the manner in which he displayed our data in the table using his reference #12, which makes our data appear to be the worst of all that quoted, which is simply not the case.

The article of ours which he quoted is 12 years out of date at present and includes mortality and morbidity figures since our study began in 1957. At that time we were operating on acute and progressing strokes and were not using shunts. This is, therefore, not a true representation of the situation as it exists today. In the last 17 years we have used general anesthesia, routine shunts, and have abandoned operation on acute and progressing strokes in a series of more than 1300 operations. Our overall operative mortality has been 1.4%, being 1.1% for TIAs and zero for asymptomatic bruits. Proper classification of patients has to be used when carotid mortality data are reported.

Likewise, in a series of 516 recent operations on patients with TIAs and asymptomatic bruits, all operated upon by the same surgeon, our total incidence of mild and severe permanent deficits has been 1.36%.

Granted that selective shunting properly used gives excellent results, we continue to use and advocate routine shunting. Dr. Ferguson states, "In fact, the best results have been reported by those who avoid shunts." He quotes Baker, et al, in this category as having a stroke rate of 1.6% with no shunting. However, in the article by Baker, et al, if one looks at the data, one sees that when operation is performed with the contralateral carotid occluded and no shunt is used, the stroke rate was 3 in 34 patients, or 8.8%. In our own series of 142 such operations done without routine shunting, there was one permanent neurologic deficit, or an incidence of 0.7%. The advocacy of no shunting at all times is unacceptable. One must know and use the proper indications for shunting when advocating selective shunting if best results are to be obtained.

Thank you very much.

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Controversy in Neurovascular Surgery


The major debate that has existed with respect to the intraoperative cerebral protection in patients undergoing carotid endarterectomy is whether one should routinely use an intraluminal shunt or selectively shunt those patients identified as having poor collateral blood flow. A new school of thought has developed, to which Dr. Ferguson subscribes, and that is based upon the hypothesis that no one requires intraoperative shunting during expedient carotid endarterectomy with the additional protection of general anesthesia. The series that we presented in our report was used to justify the concept of selective shunting as an alternative to the routine shunting based upon our results that demonstrated a combined stroke morbidity and mortality of 1.5 percent. Since we have no experience with routine non-shunting, I cannot comment on that option, nor was our report intended to imply such a position. The thesis presented in our report is simply that there are patients who do not require internal shunt. Their operation can be done safely during temporary, but unhurried crossclamping of the carotid artery. I presume the only area of dispute between Dr. Ferguson and ourselves is whether or not the other patients are or are not better off with the use of a temporary shunt. Certainly the experience with carotid endarterectomy done under local anesthesia indicates that crossclamping of the carotid artery in patients with back pressures of less than 25 mm of mercury resulted in varying degrees of neurologic dysfunction ranging from mild obtundation to hemiparesis; these deficits are promptly reversed with clamp removal or placement of a shunt. Under these circumstances, I have never had the courage to persist without a shunt, nor am I aware of any reports in the literature which described outcome under these conditions. Perhaps general anesthesia affords sufficient additional protection to permit reversability as Dr. Ferguson suggests. Yet, there must be a close correlation between clamp occlusion time and whether or not permanent neurologic damage is incurred. As Dr. Ferguson correctly points out, the majority of stroke complications are thromboembolic. These complications are often due to technical problems related to such considerations as endarterectomy end point. I would submit that in the small group of patients with low back pressure, any advantage gained by not using a shunt would be more than given up by a "hurried" endarterectomy. Our teaching of this operation emphasizes importance of doing a careful and deliberate endarterectomy, taking whatever time is necessary to accomplish a perfect technical result. Sufficient available time is provided by either good collateral circulation (back pressure greater than 25 mm of mercury) or the use of an internal shunt.

Dr. Ferguson rightly points out that a larger than usual percentage of patients were shunted because of our policy of shunting all patients who had experienced a prior cerebral infarction as an indication for operation. However, I beg to differ with him when he states that this is an unsubstantiated practice. In our second publication on back pressure,1 we observed that 17 percent of patients with prior cerebral infarction and a back pressure greater than 25 mm of mercury experienced a temporary worsening of their neurologic deficit when no shunt was used in contrast to similar patients with back pressures less than 25 mm of mercury in whom routine shunting was associated with no exacerbation of neurologic deficit. Green and Charleton,2 evaluating EEG response with carotid crossclamping noted that 31 percent of patients with prior stroke had a significant change with clamping in contrast to only a 10 percent incidence of EEG changes in those patients without a prior cerebral infarction. We have theorized that there is a zone of tissue around an area of cerebral infarction that is functional, but marginally perfused through collaterals and therefore may have a higher perfusion pressure than the area of cerebral infarction. We have theorized that there is a zone of tissue around an area of cerebral infarction that is functional, but marginally perfused through collaterals and therefore may have a higher perfusion pressure than the area of cerebral infarction. We have theorized that there is a zone of tissue around an area of cerebral infarction that is functional, but marginally perfused through collaterals and therefore may have a higher perfusion pressure than the area of cerebral infarction. We have theorized that there is a zone of tissue around an area of cerebral infarction that is functional, but marginally perfused through collaterals and therefore may have a higher perfusion pressure than the area of cerebral infarction.

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Response to Dr. Thoralf Sundt, Jr.'s opinion, and letters by Dr. Jesse Thompson and Dr. Wesley S. Moore

To the Editor:

It is a pleasure to respond to the letters written by Drs. Thompson and Moore and to the detailed commentary by Dr. Sundt provoked by the Editorial, "Intra-operative monitoring and internal shunts: are they necessary in carotid endarterectomy?" (Stroke 13: 287-289, 1982). Each of these respondents is highly regarded, knowledgeable and influential, and each has been a forceful advocate for either the use of routine shunting or selective shunting based on intra-operative monitoring during carotid endarterectomy. They have not, in my view, successfully refuted the fundamental thesis of the editorial: that, based on the available data in the literature, the use of shunting or intra-operative monitoring cannot be demonstrated to result in superior results in comparison to the use of no shunts. They do not quote superior results from their own experience, or from the literature. If they and others continue to be unable to do so, the only reasonable conclusion is that neither shunts nor monitoring are critical in this procedure. Their use appears to be a matter of personal preference, and insistence on their necessity would appear to be based on prejudgement rather than an objective evaluation of the available facts. The writers have raised a number of important points, however, that deserve further comment.

Dr. Thompson expressed concern that quoting from an outdated paper might have thrown a bad light on his results. This was not intended. His more recent reports \(^1\) were searched diligently for a clear presentation of data regarding post-operative mortality and stroke rates in the same group of patients. This information could not be found except in the 1970 report. \(^4\) For example, in the 1978 report, a procedural mortality of 1.4% in the most recent 14 year experience is quoted (table 7). Unfortunately, no data regarding the post-operative stroke rate in these patients is provided. As Dr. Thompson is a prominent proponent of routine shunting, it was felt that there was no alternative but to use the available data in developing the argument. The fact that Dr. Thompson's experience in the past 17 years is more in keeping with other reports is evidence of unquestioned competence. The data that he quotes in his letter, however, is seemingly from two different groups of patients, and is not found in the literature in the form that was required. The real issue raised by Dr. Thompson is the need for a more uniform method of reporting the results of carotid endarterectomy, which will allow an accurate comparison of the results of various workers, as we struggle to discern the truth regarding this procedure. Clear statements regarding operative stroke and mortality rates would seem to be the minimum requirement, as they embody the central issues and controversies of this procedure.

Dr. Thompson's second point, illustrated by the report of Baker et al, \(^4\) is worth consideration. In the Editorial, the overall results reported in large series were examined and the quotation from that report is accurate. Dr. Thompson is concerned that a subgroup of patients (those with contralateral carotic occlusion) are at high hemodynamic risk. Although the rate of stroke is apparently high in the series of Baker et al for this subgroup, they noted that this rate is not statistically different than for the group without occlusion. The question of specific high risk groups has not yet been systematically examined in the literature. It no doubt should be. In my experience, patients with contralateral occlusion were operated upon without the use of a shunt and excellent results obtained. \(^5\)

Dr. Moore agrees that the majority of stroke complications with carotid endarterectomy are thromboembolic in origin. As most authors report a low rate of intra-operative stroke, logic states that hemodynamically-induced stroke must be a rare event, as stated in the Editorial. Dr. Moore implies that endarterectomy without a shunt must be "hurried." This was not implied in the Editorial and is not believed to be the case. As Dr. Moore rightfully states, the key to success is careful technique, in which the necessary time to accomplish as perfect a technical result as possible is taken. In the authors' 150 consecutive cases, the average clamp time was 33 minutes (ranging from 18 to 59 minutes). \(^3\) The exaggerated emphasis on the hemodynamic risk of carotid endarterectomy has had the unhappy consequence of insufficient emphasis being placed on the importance of the surgical technique itself.

Dr. Sundt has forcefully restated and reviewed the evolution of his well known position, which must be given due consideration coming from such an authoritative worker. His opinion commends respect but it must be responded to as an expression of opinion as he does not refute successfully the fundamental thesis of the Editorial. His results are very good, but they are not significantly different than the results being routinely reported by groups who do not use shunts or monitoring. He is quite correct in stating that the conclusions in the Editorial are based on a review of the relevant literature, and personal experience. How else should one assess a controversial area? It is agreed that there are difficulties in comparing reports in the literature, as the reported details may not be comparable. This re-emphasizes the need for the most complete and detailed exposure as possible of published data. Dr. Sundt implies that the good results reported by those groups using no shunts may reflect an avoidance of high-risk cases. This argument is rejected as unsubstantiated, although it should be examined in the future. It was felt that a tabulation of 2,964 procedures without shunts from 5 different reports was probably a fair sample of all possible types of cases. Dr. Sundt is correct in stating that it is common sense to approach high-risk cases with particular caution. In passing it is to be noted that in 200 cases reported by his group in their highest risk category (grade 4), the rate of post-operative stroke was 4.5% and of post-operative death was 4.5%, in spite of the use of monitoring and selective shunts. \(^5\) One might wonder if such high-risk cases are suitable candidates for surgery at all.

There is a striking difference in the monitoring results obtained by Dr. Sundt's group and ourselves. They report clamped flows of 14 ml/100g min or less in 18.4%, flows less than 10 ml/100g min in 5.5%, and essentially zero flow in 2% of their cases. He suggests that there was a significant risk of hemodynamic infarction in all of these patients, and particularly in those with flows less than 5 ml/100g/min. But such a conclusion is completely at odds with the extensive experience of those who do not use shunts. Of the 2,964 cases in the series in which shunts were not used quoted in the Editorial, only 1% suffered post-operative stroke. Less than this percentage arose intra-operatively. Everyone appears to be in agreement that many, if not most, intra-operative strokes are thromboembolic in origin. It follows that the number of patients at hemodynamic risk must be significantly less than 1%. Our method of measuring flow is somewhat different than that used by Dr. Sundt's group, which may account for the lack of extremely low flows in our patients. We measure flow using a 10-minute clearance method mid-way through the endarterectomy, when collateral potential is likely to be fully developed. They measure flow immediately on cross-clamping, which may give spuriously low results, as there may not have been sufficient time for collateral flow to develop. The numerical accuracy of the extremely low flows reported by Sundt et al \(^6\) are highly suspect, especially if a 2-minute clearance method is used. If we agree, however, that flows less than 20 ml/100g/min are not uncommon (29% of our cases and 37% of the cases reported by Sundt et al), what is the clinical meaning of such low flows? No one but ourselves. \(^7\) Has ever tested the assumption that these flows, and the associated EEG changes, are critical. We have yet to find adverse clinical effects related to the low flows and EEG changes which occur in nearly one-third of our cases.

In stating that we find no convincing evidence for the necessity of shunts or monitoring on the basis of our experience and a critical

References
evaluation of the literature we are not taking a cavalier attitude towards this operation. The exact opposite is true. We are attempting to face the known facts. The important issue, of course, is to ensure that the real risks of this surgery are identified. There is no virtue in attempting to smite an imaginary dragon. It is obvious that the hemodynamic risks in carotid endarterectomy have been greatly exaggerated in the literature. Old assumptions need to be re-evaluated, rather than to be accepted on faith. The key to success appears to relate most to patient selection and surgical skill. It is not advocated, however, that anyone discard a practice in which they feel confident. Although in theory a few patients who are at significant hemodynamic risk might benefit from the use of a shunt, they cannot yet be accurately identified. There are potential problems with the use of a shunt, and as a result the net benefit from it's use has yet to be convincingly demonstrated.

The purpose of the Editorial was to prompt discussion (which it appears to have done) in the hope that this might help resolve the controversies regarding the safest method of performing this surgery. Time will tell if the viewpoint of the Editorial is correct.

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References

Does the Geometry of the Carotid Bifurcation Affect its Predisposition to Atheroma

To the Editor:

It has been suggested that the characteristics of flow patterns at the carotid bifurcation may have a bearing on its frequent involvement by atheroma. Flow separation occurs opposite the orifice of side branches like the external carotid artery and has been implicated in the development of plaques at the junction of the common and internal carotid arteries.1 Platelet collisions, with each other, and with the vessel wall are increased in vortices at such sites.2

It seemed possible that individual variations in the geometry of the carotid bifurcation might be relevant to the risks of development of atheroma therefore. The absolute size of the carotid vessels might also be relevant, a previous study having suggested that the smaller of the 2 internal carotid arteries was more likely to show angiographic evidence of wall disease.3

Angiograms from 150 patients were traced and the diameter of the common (CC) and internal carotid (IC) arteries measured. The maximal diameter of the sinus was recorded, and that of the internal carotid artery above the sinus. The angles between the common, internal, and external carotid (EC) arteries were taken from whichever angiographic view showed the greatest separation of the IC and EC.

As the table shows there were no significant differences in the size of the common carotid artery and the undiseased upper parts of the internal carotid artery when 48 patients with atheromatous changes at the bifurcation were compared with 102 with normal appearances. There was a reduction in the maximal diameter of the diseased segment of the internal carotid artery which may be a reflection of the difficulty in measuring the vessel width when there are plaques present.

The angle between the IC and EC was not significantly different in the two groups but there was a suggestion that the line of the common carotid and internal carotid artery was straighter in those with atheroma.

These results suggest that major differences in the geometry of the carotid bifurcation are unlikely to explain the different predisposition to carotid atheroma of different individuals. The minor changes detected seem more likely to reflect the effects of disease.

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To the Editor:

The observation that one carotid artery may have severe occlusive disease while its opposite seems uninvolved is an incontrovertible fact. A search for an explanation of this problem is clearly worthwhile for it might unlock some of the factors in the etiology of ischemic stroke. Harrison and Marshall put forth data that argues against size or angulation as important causative factors.

In our study of vessel size, we carefully chose vessels with severe obstruction (less than 2 mm residual lumen) as clearest examples of significant occlusive disease. Harrison and Marshall analyze patients with "atheromatous change" at the bifurcation and contrasted their vessel size with those with "normal appearance." It is not clear what is meant by atheromatous change. Are these shallow plaques, ulcerated plaques, or stenosis? If stenosis, to what degree?

These points are important since the capability of arteriography to differentiate small plaques from normal is questionable. Also, the relationship of that shallow plaques to stroke is uncertain. Stenosis and occlusion are undoubtedly related to stroke. When a vessel is examined directly at surgery or post-mortem, there is usually some atheromatous change, often insufficient to encroach on the lumen but making the vessel hard and yellow. Baker in his study of atherosclerosis used these atheromatous changes to study the epidemiology of atherosclerosis.

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Table

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<th>Geometry of bifurcation</th>
<th>Angiography</th>
<th>CC</th>
<th>IC</th>
<th>sinus</th>
<th>IC above</th>
<th>EC-IC</th>
<th>CC-IC</th>
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<td>(n = 102)</td>
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<td>±18.2</td>
<td>±16.5</td>
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<td>4.96</td>
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<td>(n = 48)</td>
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<td>±1.9</td>
<td>±1.03</td>
<td>±20.4</td>
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Editor’s Note: Comments on the above Dr. Harrison and Dr. Marshall letter were solicited from Dr. Louis Caplan and Dr. Frank LoGerfo. Their comments follow.
Operative mortality following carotid endarterectomy.

J E Thompson

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