Intra-Operative Monitoring and Internal Shunts: Are They Necessary in Carotid Endarterectomy?

The purpose of carotid endarterectomy is to reduce the risk of future stroke. Unless the incidence of stroke related to the surgery is extremely low, there is little likelihood of benefit for patients. It is widely held that clinically significant ischemia is a relatively common sequel to temporary carotid clamping.1,2 As a result, there has been great emphasis since the inception of carotid endarterectomy3 on the development of methods to protect the brain from ischemic insult, and to identify those patients at particular risk. The use of local or regional anaesthesia, which undoubtedly exaggerates the risk of ischemia, has given way in most clinics to the use of general anaesthesia which increases the tolerance of the brain to temporary carotid occlusion by enhancing cerebral blood flow, reducing cerebral metabolic requirements, and improving the control of arterial gas concentrations.3

However, anxiety persists regarding the possibility of intra-operative stroke directly attributable to critical reductions in flow during cross-clamping. The result is a voluminous and generally uncritical clinical literature in which prominent advocates offer contradictory advice as to the necessity and best means of affording cerebral protection by the use of intra-operative monitoring and internal shunts. These are more than minor technical matters for the surgeon, and a rational resolution of the controversies raised is highly desirable. The expectation of perfect results from carotid endarterectomy is increasingly great, and the prospect of litigation, however unjustified, is constantly present. If the likelihood of hemodynamic ischemia is not as great as generally supposed, then undue emphasis has been given to this aspect of the surgical problem and perhaps insufficient emphasis to patient selection, anaesthetic technique, and the importance of meticulous surgery. As well, certain assumptions regarding the ischemic tolerance of the brain may be brought into question. Internal shunts are not without their own risks, and may provide the surgeon with a false sense of security. Their unwarranted use will expose the patient needlessly to the risk of embolization of atheromatous debris, or intimal dissection and acute occlusion, and may limit the exposure of the plaque and the adequacy of the endarterectomy. What are surgeons and other practitioners with an interest in this procedure to think in the face of this conflicting advice regarding monitoring and shunts? Why has a consensus not been possible to date?

The report of Hunter et al in this issue of STROKE purporting to establish that carotid "back" pressure measurements are useful in determining the need for an internal shunt during carotid endarterectomy does not help in resolving the issue. They suggest that a significant proportion of patients undergoing carotid endarterectomy require a shunt to prevent post-operative neurological complications, but provide no compelling evidence for such a claim, and make no reference to an important body of literature that does not support such a contention. They argue that a "back" pressure of less than 25 mmHg (whether this is mean, systolic, or diastolic pressure is not specified) represents a critical threshold value for ischemia that demands a shunt to avoid stroke. They offer no convincing objective evaluation of this threshold value. A back pressure less...
than 25 mmHg was found in only 10 of 139 procedures, yet a shunt was used in 50% of their cases, based on the unproven proposition that any patient with a prior stroke requires a shunt regardless of the "back" pressure value. This approach confuses the issue of the importance of the back pressure measurement, the indicated purpose of their report. Their results are not exceptional. Two procedures (1.4%) resulted in an immediate stroke. One stroke, culminating in death, occurred as a direct complication of the use of a shunt that was probably unnecessary, and the other stroke occurred in a patient in whom a shunt was not used and in whom the "back" pressure was high. Their conclusion that this experience supports their criteria for shunting seems illogical, as one might argue that in one case stroke was related to the unwarranted use of a shunt, while in the other case the measurement of high "back" pressure afforded no protection.

Is there convincing evidence in the literature that the use of intra-operative monitoring and/or internal shunts lessen the risk of stroke and death in carotid endarterectomy? The table provides a selection of surgical reports organized according to whether a shunt was never used, always used, or used selectively on the basis of monitoring, and includes results from both neurosurgeons and vascular surgeons. General anesthesia was used in each series. Unfortunately, in some of the reports it is not possible to determine whether the strokes arose intra-operatively, or were delayed, occurring as a result of occlusion at the endarterectomy site or intracerebral hemorrhage. These data do not support the notion that either routine or selected use of shunts reduces the risk of stroke or death from the procedure. In fact, the best results have been reported by those who avoid shunts. The overall stroke rate in 2,964 procedures without shunts was 1.0%, and possibly no more than one-half of these strokes arose intra-operatively. This evidence indicates that significant hemodynamic ischemia during temporary carotid occlusion under general anaesthesia is a rare event.

If one sets aside, for the moment, the clinical data cited above, is there evidence that intra-operative monitoring is clinically meaningful, or that one method is superior to another? Three methods of monitoring have been most commonly discussed: (1) the measurement of carotid "stump" or "back" pressure, \(^6\) \(^7\) \(^8\) \(^9\) \(^10\) (2) continuous EEG monitoring \(^11\) \(^12\) \(^13\) \(^14\) \(^15\) \(^16\) \(^17\) \(^18\) (3) intra-operative cerebral blood flow measurements either alone or in combination with EEG monitoring. \(^19\) \(^20\) \(^21\) \(^22\) \(^23\) The criteria for using a shunt have varied somewhat, but have generally been recommended for a mean carotid "back" pressure of less than 50 mmHg, "major" changes in the EEG during cross-clamping (usually defined as the appearance of slow wave activity, suppression of greater than 50%, or both), and CBF during clamping of less than 20 ml/100g/min. No single method or combination of methods has been shown to produce clinically superior results. Carotid "back" pressure measurements have the virtue of simplicity, but their significance has been questioned. \(^24\) EEG monitoring is probably the most widely used method of intra-operative surveillance. The changes seen undoubtedly reflect a failure of neuronal electrical function. The reported frequency of these changes has varied from as low as 9.8% to as high as 29%. \(^25\) Reports on cerebral blood flow monitoring are limited as the technique has not been generally available. There has been no objective evaluation of the clinical significance of the changes noted on the EEG, or of the validity of the critical values chosen for carotid "back" pressure or CBF during carotid clamping.

A series of patients undergoing carotid endarterectomy have now been studied with intra-operative monitoring in whom no shunts were used regardless of the findings in order to evaluate the clinical consequences of the changes noted. \(^26\) The justification for this approach was the knowledge that excellent surgical results were being achieved routinely without the use of shunts. Major EEG changes during cross-clamping occurred in 35 of 102 cases. One patient suffered an intra-operative stroke associated with an EEG change, but it was not possible to determine with certainty if this had been the result of low flow or an embolus. Three patients in whom there were no EEG changes suffered minor, transient deficits, equivalent to TIA's. These were almost certainly embolic in origin. Carotid "stump" pressures were available for correlation with the EEG in 40 patients. Twenty-eight patients had mean pressures equal to or less than 50 mmHg, 15 of whom had major EEG changes and 13 of whom did not. None of the 40 patients had a post-operative deficit. While there was a trend to a greater reduction in "stump" pressure in those patients with EEG changes, in any given patient it was impossible to predict whether or not a mean pressure less than 50 mmHg would be

<table>
<thead>
<tr>
<th>Report</th>
<th>Number of operations</th>
<th>Death rate</th>
<th>Stroke rate</th>
<th>Combined rate</th>
</tr>
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<tbody>
<tr>
<td>No shunts</td>
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<td></td>
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</tr>
<tr>
<td>Baker et al(^7)</td>
<td>304</td>
<td>0.6%</td>
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<tr>
<td>Whitney et al(^8)</td>
<td>1,917</td>
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<td>0.9%*</td>
<td>2.7%</td>
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<tr>
<td>Ott et al(^9)</td>
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<td>Bland and Lazar(^10)</td>
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<td>1.1%</td>
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<td>Allen and Preziosi(^11)</td>
<td>154</td>
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<td>0.6%*</td>
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<tr>
<td>Routine shunts</td>
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<tr>
<td>Thompson et al(^12)</td>
<td>748</td>
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<td>Giannotta et al(^13)</td>
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<td>Selected shunts</td>
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<td>Ojemann et al(^1)</td>
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<td>Sundt et al(^14)</td>
<td>1,145</td>
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<td>2.0%*</td>
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\(^*\) Intra-operative strokes.
associated with an EEG change. Measurements of CBF were made in 27 patients using the intra-carotid injection of 133-Xenon. Again, the correlation between flow and EEG changes in individual cases was unreliable. Eight patients had no EEG change. Four patients with EEG change had flows above 20 ml/100g/min. None of the 27 patients had a post-operative deficit.

This review of the available information leads to the inescapable conclusion that neither intra-operative monitoring nor the use of internal shunts are necessary to avoid intra-operative stroke in carotid endarterectomy, as the usual cause for such stroke is an embolus. Although monitoring provides interesting data, the parameters cited in the literature for ischemic tolerance do not, in fact, correlate with clinically detectable post-operative deficits. This should not be totally unexpected as the situation in carotid endarterectomy is not one of arrest of cerebral circulation, but rather a relative reduction in flow. Tolerance will be a function of the extent and duration of the flow reduction. Although flow is not uncommonly reduced to a level at which reversible dysfunction of neuronal activity occurs, skillful general anesthesia together with relatively short occlusion times are sufficient to prevent failure of energy metabolism and irreversible cell damage.

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References
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