Management of Cervical Bruits and Carotid Stenosis in Preoperative Patients

ROBERT G. HART, M.D.,* AND J. DONALD EASTON, M.D.†

A PERIOPERATIVE STROKE is one which occurs intraoperatively or in the several days following an operation. It occurs in 0.3% of general surgical patients, in about 1% of patients undergoing peripheral vascular reconstruction (PVR), and in 1–5% of patients undergoing coronary artery bypass grafting (CABG).1-13

An aggressive approach to the detection, evaluation and treatment of asymptomatic cervical bruises and carotid stenosis in preoperative patients has been recommended in an effort to prevent perioperative stroke.14-16 This approach is based on the often unstated assumption that perioperative stroke is mediated by untreated carotid occlusive disease potentiated by intraoperative hypotension resulting in stroke.15-17

Although this scenario of perioperative stroke and its prevention is intuitively reasonable, justification for prophylactic carotid endarterectomy (CE) in preoperative patients requires critical review of four issues:

1. What is the prevalence of asymptomatic carotid occlusive disease in preoperative patients?
2. Can subgroups of preoperative patients with increased risk of perioperative stroke be effectively identified?
3. What are the mechanisms of perioperative stroke in these subgroups (and, consequently, is prophylactic CE likely to be effective)?
4. Is the morbidity and mortality of prophylactic CE in these patients less than the perioperative stroke risk in patients who do not undergo prophylactic CE?

Patients undergoing PVR (including abdominal aortic surgery) and CABG are often considered to be at special risk of perioperative stroke on the basis of associated atherosclerotic carotid disease. We review the predictive value of cervical bruit and stenosis for perioperative stroke in CABG and PVR patients. Potential differences in stroke prevalence and mechanism in these two groups necessitates their separate analysis. Based on aggregate data, an approach to management is suggested and the specific issues requiring further study are defined.

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PVR Patients: Cervical Bruits and Carotid Occlusive Disease

Asymptomatic cervical bruises occur in approximately 5% of the stroke-aged population and in 16–32% of patients undergoing PVR (table 1). Cervical bruises are not invariably associated with carotid occlusive disease but may arise from several sources.22,23 Further, cervical bruises may be present with hemodynamically insignificant degrees of carotid stenosis, only to disappear when the lumen is 1–2 mm or less in diameter.14,17,22

The fraction of cervical bruises in preoperative PVR patients that is associated with carotid occlusive disease is difficult to ascertain, varying with the method of investigation and criteria used to define abnormality. Noninvasive tests (NIT) of carotid occlusive disease, including oculoplethysmography (methods of Gee and Kartchner), Doppler ultrasound techniques and carotid phonoangiography, are capable of detecting stenotic lesions compromising more than 50% of the lumen diameter with 90% sensitivity in experienced hands.6,20 When carotid arteries of PVR patients with asymptomatic cervical bruises are examined by NIT, only 28–44% show occlusive disease of the carotid bifurcation (table 1). A single arteriographic study of 169 asymptomatic preoperative patients with cervical bruises (85% undergoing PVR or CABG) is available. It showed stenosis greater than 50% of the lumen diameter in 47% of arteries with bruises.22 An additional 5% of arteries with bruises showed total occlusion.22

Cervical bruises appear to be more common in preoperative PVR patients with a prior history of cerebral ischemia (table 1). This may reflect more careful cultivation of symptomatic patients. More likely it reflects the probability of cervical bruises being a marker, albeit unreliable, of cerebrovascular disease.

Prospective application of NIT to unselected, asymptomatic PVR patients reveals evidence of carotid stenosis in 13–18% (table 2). The report of Turner-seed et al. includes a large number of PVR patients with prior cerebrovascular symptoms and demonstrates a higher prevalence of cervical bruises and carotid stenosis than do reports of asymptomatic patients (table 2).6 Of arteries with carotid occlusive disease demonstrated by NIT, only 43–50% have associated cervical bruises.2,19

Cervical bruises appear to be an insensitive (about 50%) and nonspecific (about 50% false-positive rate)
CAROTID DISEASE IN PREOPERATIVE PATIENTS/Hart and Easton

**Table 1 Cervical Bruits in PVR and CABG Patients**

<table>
<thead>
<tr>
<th>Patients</th>
<th>Mean</th>
<th>Range</th>
<th>Author</th>
<th>% of patients with bruit</th>
<th>Mean</th>
<th>Range</th>
<th>Author</th>
<th>% of bruits with stenosis†</th>
</tr>
</thead>
<tbody>
<tr>
<td>PVR asymptomatic‡</td>
<td>20%</td>
<td>16–32%</td>
<td>2,6,18,19</td>
<td>38%</td>
<td>28–44%</td>
<td>2,18,19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PVR symptomatic§</td>
<td>51%</td>
<td>40–64%</td>
<td>3,4,6</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>CABG asymptomatic</td>
<td>10%</td>
<td>3–20%</td>
<td>2,6,11,12,17</td>
<td>57%</td>
<td>—</td>
<td>—</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>CABG symptomatic</td>
<td>26%</td>
<td>—</td>
<td>6</td>
<td>49%</td>
<td>45–54%</td>
<td>20,21</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixed</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>50%</td>
<td>47–53%</td>
<td>6,22</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*PVR = peripheral vascular reconstruction; CABG = coronary artery bypass grafting.  
†stenosis = > 50% carotid stenosis or occlusion by noninvasive testing or angiography.  
‡asymptomatic = no prior history of TIA or stroke.  
§mixed = prior history of TIA or stroke.  
| mixed = PVR and CABG patients that could not be separated.  

See Addendum for recently published data not included.

**PVR Patients: Perioperative Stroke Related to Cervical Bruits**

There are five reports describing the relationship of cervical bruits to perioperative stroke in PVR patients (table 3).

Individually and in aggregate, these reports conclude that cervical bruits do not effectively identify subgroups of asymptomatic PVR patients at risk for perioperative stroke. Perioperative stroke in these asymptomatic patients occurred in 1% (2/206) of patients with bruits and in 1% (9/1102) of patients without bruits (table 3).

Because cervical bruits in preoperative PVR patients correlate poorly with carotid stenosis, and perhaps even more poorly with hemodynamically significant stenosis, this lack of correlation between asymptomatic cervical bruit and perioperative stroke is not surprising. There are, however, several caveats. Three of these reports were retrospective and small numbers of patients in two reports were nonrandomly selected for prophylactic CE. Nevertheless, in the two reports of consecutive patients with no prophylactic CE, the perioperative stroke prevalence in asymptomatic PVR patients was 1% (1/115) with bruits and 1% (5/598) without bruits.

Turnipseed et al.'s data on 16 perioperative stroke patients included 10 with nonfocal, "diffuse neurologic deficits" which are difficult to compare directly to studies reporting only focal deficits.

The prevalence of perioperative stroke is increased in preoperative PVR patients with and without cervical bruits who have prior cerebral ischemia (table 3). These data further support the poor correlation between preoperative stroke and cervical bruit but suggest a subgroup of perioperative PVR patients at increased risk of perioperative stroke: no prior cerebrovascular symptoms — 1% (11/1308) stroke rate; with prior symptoms — 4% (8/226) (table 3). In contrast, in 234 patients undergoing either PVR or CABG, Kartchner and McRae reported a 4% (4/110) stroke rate in asymptomatic patients and 4% (5/124) stroke rate in patients with prior cerebrovascular symptoms.

A single report purports to dispute the lack of relationship of cervical bruit to perioperative stroke. Thompson reported 5 (4%) perioperative strokes in
138 patients with asymptomatic bruits undergoing a variety of surgical procedures. It is not stated if the study was prospective or retrospective, whether the patients were consecutive, or what the prevalence of perioperative stroke was in age-matched controls without bruits.

In summary, cervical bruits do not identify a subset of PVR patients at special risk for perioperative stroke. On balance, the literature supports Evans and Cooperman who state: “we can find no data to support asymptomatic prior cerebrovascular symptoms.”

### PVR Patients: Perioperative Stroke Related to Carotid Stenosis

There are only two studies relating perioperative stroke to carotid stenosis, as defined by NIT, in preoperative PVR patients (table 4). Barnes et al. prospectively evaluated 125 consecutive, asymptomatic PVR patients using Doppler ultrasound with spectral analysis. Perioperative stroke occurred in 1 of 23 patients with severe carotid stenosis but was contralateral to the side of stenosis, and in 1 of 102 patients without carotid stenosis. Turnipseed et al. reported 7 perioperative strokes, 5 of which were nonfocal, in 160 PVR patients of whom one-third had prior cerebrovascular symptoms: 3 strokes occurred in 62 patients with carotid stenosis (5%) versus 4 strokes in 98 patients without carotid stenosis (4%). Aggregate data combining asymptomatic and symptomatic patients from these two studies do not demonstrate an important increase in perioperative stroke in patients with carotid stenosis defined by Doppler NIT.

Conflicting data have been presented by Kartchner and McRae using oculoplethysmography (OPG). Of 234 patients undergoing PVR or CABG, the perioperative stroke rate was 1.2% (2/192) in patients with normal OPG and 17% (7/42) in those with OPG evidence of moderate flow reduction (> 30% flow reduction, > 60% diameter stenosis). There were 31 patients who had initially abnormal OPG and who were nonrandomly selected for prophylactic CE among the group with the 1% stroke rate. It was not stated how the patients were chosen for OPG or the relationship of the stroke to the side of OPG abnormality of timing of surgery, and 53% of the patients had experienced prior cerebrovascular symptoms.

A possible explanation for the reported differences in perioperative stroke rate associated with abnormal Doppler NIT versus normal OPG could be the relative specificity for high degrees of stenosis. If, for example, abnormal OPG reflected higher degrees of stenosis (estimated to be > 60% diameter reduction by the authors) than abnormal Doppler studies (>

### Table 3: Cervical Bruits and Perioperative Stroke

<table>
<thead>
<tr>
<th>Author</th>
<th>Patient group</th>
<th>With bruit</th>
<th>No bruit</th>
</tr>
</thead>
<tbody>
<tr>
<td>PVR*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Turnipseed et al</td>
<td>mixed</td>
<td>6% (n = 70)</td>
<td>3% (n = 90)</td>
</tr>
<tr>
<td>Barnes et al</td>
<td>asymptomatic</td>
<td>4% (n = 23)</td>
<td>1% (n = 102)</td>
</tr>
<tr>
<td>Treiman et al</td>
<td>asymptomatic</td>
<td>2% (n = 63)</td>
<td>1% (n = 369)</td>
</tr>
<tr>
<td>Treiman et al</td>
<td>symptomatic</td>
<td>0% (n = 21)</td>
<td>0% (n = 36)</td>
</tr>
<tr>
<td>Carney et al</td>
<td>asymptomatic</td>
<td>0% (n = 28)</td>
<td>1% (n = 135)</td>
</tr>
<tr>
<td>Carney et al</td>
<td>symptomatic</td>
<td>0% (n = 5)</td>
<td>25% (n = 4)</td>
</tr>
<tr>
<td>Evans &amp; Cooperman</td>
<td>asymptomatic</td>
<td>0% (n = 92)</td>
<td>1% (n = 496)</td>
</tr>
<tr>
<td>Aggregate</td>
<td>asymptomatic</td>
<td>1% (n = 206)</td>
<td>1% (n = 1102)</td>
</tr>
<tr>
<td></td>
<td>symptomatic/mixed</td>
<td>4% (n = 96)</td>
<td>3% (n = 130)</td>
</tr>
<tr>
<td>CABG*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Martin &amp; Hashimoto</td>
<td>mixed</td>
<td>14% (n = 7)</td>
<td>3% (n = 245)</td>
</tr>
<tr>
<td>Turnipseed et al</td>
<td>mixed</td>
<td>7% (n = 28)</td>
<td>5% (n = 142)</td>
</tr>
<tr>
<td>Barnes et al</td>
<td>asymptomatic</td>
<td>10% (n = 21)</td>
<td>1% (n = 303)</td>
</tr>
<tr>
<td>Breslau et al</td>
<td>asymptomatic</td>
<td>0% (n = 18)</td>
<td>3% (n = 72)</td>
</tr>
<tr>
<td>Aggregate</td>
<td>asymptomatic</td>
<td>5% (n = 39)</td>
<td>2% (n = 375)</td>
</tr>
<tr>
<td></td>
<td>mixed</td>
<td>9% (n = 35)</td>
<td>4% (n = 387)</td>
</tr>
<tr>
<td>General surgical</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thompson</td>
<td>unknown</td>
<td>4% (n = 138)</td>
<td>—</td>
</tr>
<tr>
<td>Dorazio et al</td>
<td>asymptomatic</td>
<td>0% (n = 53)</td>
<td>—</td>
</tr>
</tbody>
</table>

*PVR = peripheral vascular reconstruction; CABG = coronary artery bypass grafting.
†mixed = combination of asymptomatic and symptomatic patients.
‡asymptomatic = no prior cerebrovascular symptoms.
§symptomatic = prior history of TIA or stroke.
\*most events in Turnipseed et al were nonfocal.
See Addendum for recently published data that is not included.
Mechanisms of Perioperative Cerebral Infarction

The mechanism of perioperative cerebral infarction is often uncertain. A review of this complication in PVR patients reported in the literature shows the stroke onset to be postoperative, not intraoperative, in all thirteen patients in whom the temporal profile is stated. In 38 CABG patients with focal perioperative stroke, 16 (42%) had the onset of their neurologic deficit postoperatively.9,12 Thus, mechanisms other than intraoperative hypotension potentiating carotid occlusive disease must be etiologic in some portion of perioperative stroke patients.9,11

Comparison of the prevalence of cervical bruises and carotid stenosis in patients with and without perioperative stroke suggests a role for cerebrovascular disease in perioperative stroke (table 5). Although only small numbers of patients are available for comparison, aggregate data show an increase in cervical bruises in perioperative stroke patients (28%) versus non-stroke patients (13%). Similarly, carotid stenosis demonstrated by NIT is more prevalent in patients with perioperative stroke (40% versus 16%) (table 5). Nevertheless, it is clear from Tables 3 and 4 that the predictive value of carotid bruises and stenosis by NIT is minimal, partly due to the very low perioperative stroke rate even in the presence of a bruit or stenosis.

Most reports of perioperative stroke do not define the temporal relationship of stroke to the surgery and do not consider the multiple possible mechanisms. It is possible that a subset of perioperative stroke pa-

**OPG** = oculoplethysmography.
Patients exist in whom severe carotid occlusive disease combined with intraoperative hypotension are the crucial determinants of cerebral ischemia. However, most perioperative strokes occur in the absence of carotid occlusive disease (detected by NIT) or have their onset in the postoperative period.  

Correlation of occlusive disease with more subtle forms of perioperative cerebral ischemia has not been attempted but is of potential importance.

**Perioperative Stroke: Bilateral Stenosis and Angiographic Correlations**

Bilateral carotid stenosis detected by noninvasive testing did not predict an increased risk of perioperative stroke in Barnes et al.’s report (1/25 with bilateral stenosis had stroke versus 1/38 with unilateral stenosis), and this observation was confirmed by Breslau et al. in a small number of patients. Noninvasive techniques used in the cited studies are often unreliable in predicting high degrees of stenosis/occlusion, stenosis in distal cerebral vessels and the status of potential collateral vessels. Very high degrees of stenosis may be required to reduce flow. Are there subgroups of patients with asymptomatic carotid stenosis with particular combinations of high-grade, angiographically-defined occlusive disease who are at special risk for intraoperative stroke? For example, Sweeney et al. reported ischemic optic neuropathy occurring during CABG with intraoperative hypotension in a 60-year-old man with bilateral carotid occlusions. Nevertheless, we have recently evaluated two patients who anecdotally demonstrate that even angiographic evidence of severe occlusive disease cannot be readily related to perioperative stroke risk.

**Patient #1**

Patient #1 was a healthy 83-year-old man who recently had experienced two left-hemispheric TIAs. Angiography demonstrated an isolated, high-grade (90%) stenosis of the left carotid siphon, minimal irregularity of the internal carotid bifurcations and no other abnormalities. The left middle cerebral artery showed delayed filling on the left carotid injection and was not visualized on the right carotid injection (which filled both anterior cerebral arteries) or vertebral injection. He was randomized to the medical arm of the Collaborative Extracranial/Intracranial Bypass Study. Two months later, he presented with acute bowel obstruction and underwent urgent colostomy under general anesthesia for diverticulitis-related obstruction. During the three-hour procedure, blood pressure declined from 170/80 mm Hg to 110/50 mm Hg despite special efforts to avoid hypotension. Neurological examination postoperatively was normal.

**Patient #2**

Patient #2 was a 65-year-old man with a left hemispheric TIA on the day of admission. Cerebral angiography showed total occlusion of the left internal carotid artery at the common carotid bifurcation with the left middle cerebral and anterior cerebral vessels supplied by ocular collaterals. There was only minimal irregularity of the right carotid bifurcation and no distal lesions. Twelve days after admission, he underwent aorto-femoral bypass under nitrous oxide and enflurane anesthesia, during which the blood pressure declined from 200/90 mm Hg to 120 mm Hg systolic. His immediate postoperative course was uncomplicated.

On the third postoperative day, an increased blood pressure was treated with I.V. hydralazine with subsequent fall to 150/76 mm Hg. He developed a right hemiparesis and nonfluent dysphasia which fluctuated over 3 hours before finally stabilizing.
total ipsilateral occlusion in patient #2, combined with intraoperative hypotension, there was no intraoperative stroke. A fall in cerebral metabolic rate during general anesthesia could have been protective by decreasing oxygen requirements.27 Infarction was precipitated postoperatively by less pronounced hypotension in patient #2.

Treiman et al. reported five patients with angiographically significant carotid stenosis, one with bilateral occlusions, who underwent urgent aortic aneurysm repair without prophylactic CE.3 None suffered intraoperative stroke, although one patient with bilateral stenosis developed a hemiparesis with onset on the 4th postoperative day.

We and others24, 27, 28 have observed patients with perioperative stroke in the absence of carotid occlusive disease, proven angiographically or at postmortem examination, showing that occult stenosis (false-negative NIT) is not a tenable explanation. Based on these anecdotal patients, there is no readily apparent correlation between angiographic evidence of tight carotid stenosis/occlusion and perioperative stroke.3, 33

Further, standard intra-arterial angiography is associated with a morbidity rate which is at least 1–2% in older patients with cardiovascular disease.34, 35 The application of digital subtraction intravenous angiography may allow safe, prospective definition of cerebrovascular anatomy in preoperative patients and clarify many of these issues.

Prevention of Perioperative Stroke by Endarterectomy

Since many perioperative stroke occur postoperatively and are related to embolic mechanisms, it cannot be assumed a priori that removal of a carotid stenosis will be beneficial.11, 27 Aggregate data concerning 61 patients undergoing PVR following successful prophylactic CE show no perioperative stroke.3, 4, 19, 36 (predicted stroke rate in patients with carotid stenosis from other studies is 5% (table 4)). Fields reported that perioperative stroke in patients with asymptomatic bruits was reduced from 3–5% to an “almost negligible morbidity” after angiography and endarterectomy were routinely performed in such patients.14

In patients who underwent first prophylactic CE, then CABG, the aggregate data concerning 97 procedures show one stroke and two TIAs following CABG.21, 37 Hertzler et al. reported a patient with perioperative right hemispheric TIA during CABG that followed prophylactic right CE.7 The role of prophylactic CE in stroke prevention when performed simultaneously with CABG cannot be separately assessed from the overall neurological morbidity of the combined CE/CABG procedure (5–8%).47

Kartchner and McRae reported prophylactic CE in 31 patients with abnormal OPG as part of a larger group with a perioperative stroke rate of 1% (the stroke rate in this subgroup of 31 patients could not be determined from published data). This compares to a 17% stroke rate in 41 patients with abnormal OPG who did not receive surgery. However, patient selection for OPG and surgery and the relationship of perioperative stroke to side of OPG abnormality were not defined.15 In all of the above studies, nonconsecutive and nonrandom patients were selected for prophylactic CE.

Risk of Prophylactic Carotid Endarterectomy

The potential value of prophylactic CE in asymptomatic PVR patients requires consideration of the morbidity and mortality of the procedure in this patient population. Coexistent coronary artery disease is a major cause of perioperative morbidity in PVR and makes the risk of prophylactic CE in PVR patients higher than in the general population. A review of four surgical series comprising 63 PVR patients undergoing CE, including some with prior TIA, shows a perioperative stroke rate of 5% (range 0–10%) and a mortality rate of 2%.3, 4, 19, 36 For prophylactic CE to be of value, a subgroup of PVR patients must be identified who have a natural history of perioperative stroke higher than the CE complication rate. This also assumes that CE will prevent most perioperative strokes. The long-term, potential benefit of CE in PVR patients may be considered but this seems to be a distinct issue, not involved in the decision to perform CE before other surgery.2, 29

Similarly, prophylactic CE in CABG shows a 4% (range 3–6%) risk of perioperative cerebral ischemia and an additional 3% mortality in 97 patients operated prior to their CABG.7, 21, 37 and a 5% (range 0–12%) stroke rate in 490 patients whose CE was performed simultaneously with their CABG.7, 21, 37, 46 Patients undergoing simultaneous CE/CABG who had bilateral stenosis were at special risk of complication.7, 47 These are aggregate data from reports of nonrandomized, nonconsecutive patients. It is possible that, on average, patients undergoing prophylactic CE have higher degrees of carotid stenosis than consecutive patients with carotid stenosis identified prospectively by NIT, confounding direct comparison.2, 6, 7, 44

The management options based on this review of the literature are summarized in table 6. It is emphasized that prophylactic CE patients were not chosen consecutively or randomly in these series and that they consist of patients with and without prior cerebrovascular symptoms. Only the report of Kartchner and McRae demonstrating a 17% perioperative stroke rate in selected PVR and CABG patients with abnormal OPG suggest a subgroup that could potentially benefit from prophylactic CE.15 Their data conflict with consecutive series of other authors.2, 6, 20

Conclusions

1) The prevalence of asymptomatic cervical bruits is about 20% in preoperative peripheral vascular reconstruction (PVR) patients and about 10% in preoperative coronary artery bypass grafting (CABG) patients (table 1). 2) The prevalence of asymptomatic carotid stenosis by noninvasive testing (NIT) is about 15% in preoperative PVR patients and about 10% in preoperative CABG patients (table 2). 3) Cervical bruits and carotid stenosis by NIT are more common in preoperative patients with prior cerebral ischemia (tables 1 and 2). 4) Cervical bruits are not reliable predictors of
carotid stenosis in preoperative PVR and CABG patients: about half of arteries with cervical bruits will have stenosis (table 1) and half or less of arteries with carotid stenosis by NIT will have associated bruits. 5) Neither cervical bruits or carotid stenosis demonstrated by Doppler NIT identify subgroups of asymptomatic PVR or CABG patients at substantially increased risk of perioperative stroke (tables 3 and 4): small increases (0–3%) in perioperative stroke are found in asymptomatic PVR and CABG patients with bruits and stenosis, and a single report using OPG shows an increase in perioperative stroke from 1% in patients with normal OPG to 7% in patients with abnormal OPG. 6) Cervical bruits and carotid stenosis are consistently more common in PVR and CABG patients with perioperative stroke than in patients who do not experience stroke (table 5). While cerebrovascular disease manifested by bruits and abnormal NIT is probably etiologic in many patients with perioperative stroke, it is still a poor predictor of perioperative stroke. This apparent paradox is best explained by the low prevalence of stroke even in patients who have carotid occlusive disease. 7) Multiple mechanisms are involved in perioperative stroke. Many perioperative strokes occur in the postoperative period, unrelated to intraoperative hypotension. 8) There are no series reporting the perioperative stroke risk associated with angiographically-defined carotid occlusive disease. Anecdotal reports show no consistent relationship between angiographically-defined, high-grade stenosis/occlusion and perioperative stroke. 9) There is little substantive evidence indicating that prophylactic carotid endarterectomy is of overall benefit in preoperative patients with asymptomatic carotid occlusive disease. Further information is needed before this surgery can be routinely recommended for asymptomatic preoperative patients.

**Addendum**

Since preparation of this review Ropper et al. have prospectively studied the relationship of perioperative stroke (occurring within three days of surgery) to the presence of cervical bruit in 735 unselected patients undergoing elective surgery (8% had prior cerebrovascular symptoms). Their observations included:

1) In 568 patients undergoing nonvascular procedures, 67 patients (12%) had a cervical bruit. No perioperative stroke occurred in any patient.

2) In 167 patients undergoing vascular procedures (including CABG), 37 patients (22%) had a cervical bruit. One perioperative stroke occurred ipsilateral to a symptomatic bruit (3%) and four perioperative strokes occurred in patients without bruits (3%).

3) All five perioperative strokes occurred in CABG patients and were felt to be embolic in nature. Three of these five patients had onset of deficit after awakening from anesthesia.

These prospective data strengthen the conclusions regarding the poor predictive value of cervical bruit for perioperative stroke and further question the role of intraoperative hypotension combined with carotid occlusive disease in the genesis of perioperative stroke.

**References**


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**Table 6 Outcome of Management Options**

<table>
<thead>
<tr>
<th>Natural history†</th>
<th>Author</th>
<th>n</th>
<th>% Experiencing ischemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>PVR†</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>bruits</td>
<td>2–6</td>
<td>302</td>
<td>2%</td>
</tr>
<tr>
<td>no bruits</td>
<td>2–6</td>
<td>1232</td>
<td>1%</td>
</tr>
<tr>
<td>(+) NIT§</td>
<td>2, 6</td>
<td>85</td>
<td>5%</td>
</tr>
<tr>
<td>(−) NIT</td>
<td>2, 6</td>
<td>200</td>
<td>3%</td>
</tr>
<tr>
<td>CABG†</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>bruits</td>
<td>2, 6, 20, 24</td>
<td>74</td>
<td>7%</td>
</tr>
<tr>
<td>no bruits</td>
<td>2, 6, 20, 24</td>
<td>762</td>
<td>3%</td>
</tr>
<tr>
<td>(+) NIT</td>
<td>2, 6, 20</td>
<td>72</td>
<td>4%</td>
</tr>
<tr>
<td>(−) NIT</td>
<td>2, 6, 20</td>
<td>524</td>
<td>3%</td>
</tr>
</tbody>
</table>

§(+ )NIT = abnormal noninvasive testing by Doppler methods.
| (−) NIT | normal noninvasive testing by Doppler methods.
| CABG — before | 7, 21, 37 | 97 | 4% (plus 3% mortality) |
| CABG — simultaneous | 7, 21, 37–46 | 490 | 5% |

*primarily asymptomatic patients (tables 3 and 4) plus data from 2 studies 6, 8 in which a minority of patients had prior TIA.
†Kartchner and McRae’s data (table 4) not included because of mixed CABG and PVR patients.
‡PVR = peripheral vascular reconstruction.
§(+ )NIT = abnormal noninvasive testing by Doppler methods.
(−) NIT = normal noninvasive testing by Doppler methods.
※CABG = coronary artery bypass grafting.
**nonconsecutive, nonrandom selection of patients for prophylactic surgery in all series.

See Addendum for recently published data that is not included.
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