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 bruits 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.

From the *Comprehensive Stroke Center of Oregon, Oregon Health Sciences University, Portland, Oregon and †the Department of Medicine, University of Texas Health Sciences Center at San Antonio. Address correspondence to: Dr. J. Donald Easton, Professor and Chief of Neurology, Department of Medicine, University of Texas Health Sciences Center at San Antonio, San Antonio, Texas 78284.

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TABLE 1  Cervical Bruits in PVR and CABG Patients*

<table>
<thead>
<tr>
<th>Patients</th>
<th>% of patients with bruit</th>
<th>% of patients with stenosis†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>Range</td>
<td>Author</td>
</tr>
<tr>
<td>PVR asymptomatic†</td>
<td>20%</td>
<td>16–32%</td>
</tr>
<tr>
<td>PVR symptomatic§</td>
<td>51%</td>
<td>40–64%</td>
</tr>
<tr>
<td>CABG asymptomatic</td>
<td>10%</td>
<td>3–20%</td>
</tr>
<tr>
<td>CABG symptomatic</td>
<td>26%</td>
<td>—</td>
</tr>
<tr>
<td>Mixed</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.
§symptomatic = prior history of TIA or stroke.
| mixed = PVR and CABG patients that could not be separated.

See Addendum for recently published data not included.

indicates carotid occlusive disease in preoperative PVR patients.

CABG Patients: Cervical Bruits and Carotid Stenosis

Asymptomatic cervical bruits are present in about 10% of preoperative CABG patients (table 1) and about half of such bruits are associated with carotid stenosis using NIT.2,20 One arteriographic study of 100 CABG patients with cervical bruits, of whom 37% had prior cerebrovascular symptoms, showed carotid stenosis in 45%.21

Prospective evaluation of CABG patients with various noninvasive techniques has shown evidence of asymptomatic stenosis in 6–12%, of which 30% had an associated bruit (table 2).

The prevalence of both cervical bruits and carotid stenosis by NIT is consistently less in CABG patients than in PVR patients (tables 1 and 2).18

TABLE 2  Carotid Stenosis in PVR and CABG Patients*

<table>
<thead>
<tr>
<th>Author</th>
<th>Patient group</th>
<th>N</th>
<th>Method</th>
<th>% with stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>PVR</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Barnes et al²</td>
<td>asymptomatic†</td>
<td>125</td>
<td>Doppler</td>
<td>18%</td>
</tr>
<tr>
<td>Jain et al¹⁹</td>
<td>asymptomatic</td>
<td>75</td>
<td>OPG‡</td>
<td>13%</td>
</tr>
<tr>
<td>Turnipseed et al⁶</td>
<td>mixed§</td>
<td>160</td>
<td>Doppler</td>
<td>39%</td>
</tr>
<tr>
<td>CABG</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Barnes et al²</td>
<td>asymptomatic</td>
<td>324</td>
<td>Doppler</td>
<td>12%</td>
</tr>
<tr>
<td>Breslau et al²⁰</td>
<td>asymptomatic</td>
<td>78</td>
<td>Doppler</td>
<td>6%</td>
</tr>
<tr>
<td>Turnipseed et al⁶</td>
<td>mixed</td>
<td>170</td>
<td>Doppler</td>
<td>8%</td>
</tr>
<tr>
<td>PVR/CABG</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kartchner and McRae¹⁵</td>
<td>mixed</td>
<td>234</td>
<td>OPG</td>
<td>31%</td>
</tr>
</tbody>
</table>

*PVR = peripheral vascular reconstruction; CABG = coronary artery bypass grafting.
†asymptomatic = no prior TIA or stroke.
‡OPG = oculoplethysmography.
§mixed = patients with and without prior cerebrovascular symptoms.
| mixed = comprised of PVR and CABG patients that could not be separated.

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).2,4 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 retrospective3–5 and small numbers of patients in two reports were nonrandomly selected for prophylactic CE.3,4 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.2,4 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.6

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.15

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 the necessity for preoperative carotid arteriography to determine need for prophylactic surgery." The majority of the data comes from papers that used Doppler ultrasound with spectral analysis of Doppler NIT and OPG. Abnormal Doppler NIT was used as the indicator of flow reduction in 234 patients; the perioperative stroke rate was 1% (2/234) in patients with normal Doppler NIT and 17% (4/23) in patients with abnormal Doppler NIT. The stroke rate was 0% (0/1102) in patients with normal Doppler OPG and 9% (4/42) in patients with abnormal Doppler OPG. Abnormal OPG was defined as Doppler NIT > 15 mm/s.

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% (2/234) in patients with normal OPG and 17% (4/23) 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 abnormal 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 (>...
50% stenosis), 2, 6 these differences would be compatible with a threshold phenomenon based on differences in technique. However, the overall prevalence of abnormal OPG in Kartchner and McRae’s series (31%) is higher than the prevalence of abnormal findings using Doppler NIT (15–23%). 2, 6 This suggests either that lesser degrees of stenosis were detected using OPG or that a selected patient population was studied. 15

The lack of correlation of abnormalities detected by NIT with perioperative stroke must be restricted to Doppler ultrasound techniques until further, complete information is available for OPG. Ultimately, the use of intravenous or standard angiography may offer the most useful data on the role of carotid occlusive disease in perioperative stroke in PVR patients.

### CABG Patients: Perioperative Stroke Related to Cervical Bruits

Aggregate data from four reports show an increase of perioperative stroke from 3% in patients without bruits to 7% in patients with bruits (table 3). In all asymptomatic patients, the perioperative stroke rate with CABG is 2% compared to 1% with asymptomatic PVR (table 3), despite a higher prevalence of cervical bruits and carotid stenosis in the latter group (tables 1 and 2). 2, 6

Cervical bruits do not effectively identify subgroups of CABG patients with clinically important increased risk of perioperative stroke.

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

Aggregate data from three reports using Doppler techniques show a slight increase (1.5% absolute) in perioperative stroke rate in CABG patients with carotid stenosis (more than 50% compromise of lumen diameter detected by NIT) (table 4). As noted above, Kartchner and McRae’s data using OPG in a combined population of CABG and PVR patients showed a substantial increase in perioperative stroke in patients with abnormal OPG (table 4). 15 Details of the patient population (e.g., whether consecutive, relationship of stroke to side of OPG abnormality) were not provided. 15

#### Mechanisms of Perioperative Cerebral Infarction

The mechanism of perioperative cerebral infarction is often uncertain. 11, 27 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. 2–4, 27, 38 Stroke is uncommon even in the presence of intraoperative hypotension. 6, 37 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, 27

Comparison of the prevalence of cervical bruits 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 bruits 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 bruits 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. 11, 27 It is possible that a subset of perioperative stroke pa-

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**Table 4 Carotid Stenosis and Perioperative Stroke**

<table>
<thead>
<tr>
<th>Author</th>
<th>Patient group</th>
<th>Method</th>
<th>Stroke rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>With stenosis</td>
<td>Without stenosis</td>
</tr>
<tr>
<td>PVR*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Turnipseed et al 6‡§</td>
<td>mixed§</td>
<td>Doppler</td>
<td>5% (n = 62)</td>
</tr>
<tr>
<td>Barnes et al 2</td>
<td>asymptomatic</td>
<td>Doppler</td>
<td>4% (n = 23)</td>
</tr>
<tr>
<td>Aggregate</td>
<td>all patients</td>
<td>Doppler</td>
<td>5% (4/85)</td>
</tr>
<tr>
<td>CABG*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Turnipseed et al 6‡§</td>
<td>mixed</td>
<td>Doppler</td>
<td>14% (n = 14)</td>
</tr>
<tr>
<td>Barnes et al 2</td>
<td>asymptomatic</td>
<td>Doppler</td>
<td>3% (n = 40)</td>
</tr>
<tr>
<td>Breslau et al 20</td>
<td>mixed</td>
<td>Doppler</td>
<td>0% (n = 18)</td>
</tr>
<tr>
<td>Aggregate</td>
<td>all patients</td>
<td>Doppler</td>
<td>4% (3/72)</td>
</tr>
<tr>
<td>CABG/CABG</td>
<td></td>
<td>Doppler</td>
<td>17% (n = 42)</td>
</tr>
</tbody>
</table>

*PVR = peripheral vascular reconstruction; CABG = coronary artery bypass grafting.
†carotid stenosis implies more than 50% reduction in lumen diameter.
‡most strokes in this series were nonfocal, diffuse deficits.
§mixed = patients with and without prior cerebrovascular symptoms.
| asymptomatic = no prior cerebrovascular symptoms.
| mixed§ = patients who underwent prophylactic endarterectomy.
**OPG = oculoplethysmography.

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**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. 2–4, 27, 38 Stroke is uncommon even in the presence of intraoperative hypotension. In 38 CABG patients with focal perioperative stroke, 16 (42%) had the onset of their neurologic deficit postoperatively. Thus, mechanisms other than intraoperative hypotension potentiating carotid occlusive disease must be etiologic in some portion of perioperative stroke patients. 9–12

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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-
tients 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.2,4,12-27,29

Correlation of occlusive disease with more subtle forms of perioperative cerebral ischemia has not been attempted but is of potential importance.30

### 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.1’s report (1/25 with bilateral stenosis had stroke versus 1/38 with unilateral stenosis),2 and this observation was confirmed by Breslau et al.20 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.21 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.32 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, during which the 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.

Despite high-grade, distal stenosis in patient #1 and
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 stroke3, 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 TIs following CABG.27 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.19 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.19

Prophylactic endarterectomy**

PVR

<table>
<thead>
<tr>
<th>Natural history†</th>
<th>Author</th>
<th>n</th>
<th>% Experiencing ischemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>PVR+ 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+ 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>
<tr>
<td>CABG — before</td>
<td>7, 21, 37</td>
<td>97</td>
<td>4% (plus 3% mortality)</td>
</tr>
<tr>
<td>CABG — simulta­neous 7, 21, 37–46</td>
<td>490</td>
<td>5%</td>
<td></td>
</tr>
</tbody>
</table>

*primarily asymptomatic patients (tables 3 and 4) plus data from 2 studies 20 in which a minority of patients had prior TIA. 1Kartchner and McRae’s data (table 4) not included because of mixed CABG and PVR patients. 3PVR = peripheral vascular reconstruction. 8(+) NIT = abnormal noninvasive testing by Doppler methods. 7(−) NIT = normal noninvasive testing by Doppler methods. ICABG = 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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CAROTID DISEASE IN PREOPERATIVE PATIENTS/Hart and Easton 297

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Management of cervical bruits and carotid stenosis in preoperative patients.
R G Hart and J D Easton

Stroke. 1983;14:290-297
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