Asymptomatic Carotid Bruit and Stenosis: A Reappraisal

Frank M. Yatsu, M.D. and Robert G. Hart, M.D.

NO PROBLEM in stroke management provokes more debate, uncertainty, and anxiety than asymptomatic carotid bruit or stenosis. Since atheromatous lesions at the carotid bifurcation are responsible for a sizable proportion of ischemic strokes, prophylactic extirpation of potentially offending lesions has intuitive appeal. However, after years of controversy, aggregate data do not convincingly demonstrate benefit from prophylactic surgery in asymptomatic carotid disease.

To assist clinicians in analyzing currently available data, this paper will review in detail: (1) the natural history of asymptomatic bruits in the general population, (2) the natural history of asymptomatic bruits and stenosis in referral patients or patients with other symptomatic vascular disease, (3) the natural history of asymptomatic stenosis contralateral to a symptomatic carotid artery, and (4) the predictive value of asymptomatic bruits and stenosis for perioperative stroke in patients undergoing cardiac and peripheral vascular procedures. In considering data from several studies, it is critical that the specific patient populations and the clinical methodologies be carefully analyzed to yield meaningful comparison.

**Bruits in the General Population**

Two important population-based, epidemiological studies assess the occurrence and implications of asymptomatic cervical bruits. The Framingham Study followed 171 people (66 men and 105 women) with newly detected, asymptomatic bruits for up to eight years. The prevalence of bruits increased both with age (3.5% in 44 to 54 year olds, 7.0% in 65 to 79 year olds) and with the presence of hypertension and diabetes mellitus. The stroke rate in the patients with bruits was more than twice the expected incidence, but half were either nonischemic strokes, not appropriate to the side of bruit, or due to cardiogenic embolism (Table 1).

In the rural Evans County, Georgia study, Heyman et al. followed 1,620 adults over age 45 without prior cerebrovascular or cardiac symptoms for six years. There were 72 people (4.4%) with cervical bruits, with prevalence directly correlating with age, presence of hypertension, and female sex. The presence of cervical bruits did not correlate with ischemic, ipsilateral stroke but, more reliably, signaled risk of coronary artery disease.

These prospective, population-based studies do not demonstrate clinically important increases in ipsilateral ischemic stroke associated with asymptomatic carotid bruits. There are several explanations for this poor correlation. Bruits alone may be from noncarotid sources and do not reflect the degree of stenosis, the presence of ulceration, or the status of collateral circulation, nor do they provide clues to possible platelet and coagulation abnormalities — all potentially important in stroke pathogenesis.

**Bruits in a Referral Population**

Patients referred to specialists for asymptomatic bruits, however, may represent a select population with a different “natural history.” For example, many have other vascular or systemic diseases or symptoms, such as vague cerebral dysfunction, which prompted initial medical attention. This inhomogeneity is reflected in outcome differences in the three available studies. The population of Thompson et al. was made up of unoperated patients, chosen nonrandomly for unspecified reasons, and included all types and location of stroke without regard to site of bruit. This selection bias is reflected in the particularly poor prognosis, which was even worse than the prognosis from many reports of symptomatic bruits. Ten per cent of Kachtcher and McRae’s patients were nonrandomly selected for prophylactic endarterectomy, which potentially alters the “natural history” as reflected in their data (Table 1).

In aggregated studies of referral bruits, the incidence of cerebrovascular symptoms (TIA or stroke) is about 5% yearly, but the “appropriateness” of these symptoms is often not stated.

**Bruits in Preoperative Patients: Long-Term Outcome**

The long-term (not perioperative) outcome of asymptomatic bruits detected in preoperative patients undergoing major peripheral vascular and coronary artery bypass procedures shows a higher incidence of cerebral ischemia than bruits detected in population-based, epidemiological surveys (Table 1). This undoubtedly reflects a greater severity of generalized atherosclerosis; fully 10 to 20% of these patients harbor asymptomatic bruits. However, associated coronary artery disease places these patients at potentially greater risk for complications of prophylactic carotid surgery (perioperative myocardial infarction) includ-
ing perioperative stroke because of more severe, generalized cerebrovascular disease compromising collaterals. Ischemic infarction incidence is about 3% yearly in these patients with perhaps an additional 5% experiencing TIA. The very high figure for TIA in the study of Barnes et al. may reflect the brief (11-month) follow-up with high early stroke incidence (table 1).

**Bruits and Stenosis in Preoperative Patients: Perioperative Stroke**

It is a reasonable concern that intraoperative hypotension may complicate asymptomatic carotid occlusive disease by causing perioperative strokes during major vascular procedures, including coronary artery bypass surgery. However, the seven reports addressing this problem disclose no clinically important increase in perioperative strokes (table 2). However, Kartchner & McRae have reported divergent data: Of 234 patients undergoing major vascular procedures, perioperative strokes occurred in 1% of those with normal and 17% of those with abnormal oculoplethysmography. Details of whether these strokes were in consecutive, previously asymptomatic patients and of stroke location, variety, and timing were not given.

Correlation of asymptomatic carotid disease with more subtle postoperative neurological deficits has not been attempted but is of potential importance.

**Asymptomatic Stenosis Detected Noninvasively**

Since cervical bruises are relatively insensitive and nonspecific indicators of carotid occlusive disease (only 10% of all strokes in the Framingham Study had associated ipsilateral bruises), attention has been directed to detection of carotid stenosis by various types of noninvasive tests (NIT), including Doppler ultrasound, oculoplethysmography, and real-time Doppler imaging. When patients with asymptomatic bruits are evaluated with these techniques, only 15 to 50% have asymptomatic stenosis detected by these tests.

*References 13, 14 and 23 include a small number of patients with prior cerebrovascular symptoms.

**Table 1** Asymptomatic Cervical Bruits and Cerebral Ischemia*

<table>
<thead>
<tr>
<th>Population-based</th>
<th>Mean follow-up</th>
<th>With bruits</th>
<th>No bruits</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Wolf et al.</strong> (Framingham)</td>
<td>4yr</td>
<td>171</td>
<td>3%† (1%)</td>
</tr>
<tr>
<td><strong>Heyman et al.</strong> (Evans County)</td>
<td>6yr</td>
<td>72</td>
<td>2.3% (?)</td>
</tr>
<tr>
<td><strong>Kagan et al.</strong> (Honolulu)</td>
<td>4yr</td>
<td>124</td>
<td>1% (?)</td>
</tr>
<tr>
<td><strong>Referral-based</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Dorazio et al.</strong></td>
<td>7yr</td>
<td>97</td>
<td>2.7%(1.6%)</td>
</tr>
<tr>
<td><strong>Kartchner and McRae</strong></td>
<td>2yr</td>
<td>1130</td>
<td>1.5%(1.5%)</td>
</tr>
<tr>
<td><strong>Thompson et al.</strong></td>
<td>4yr</td>
<td>138</td>
<td>4.5%(6.5%)</td>
</tr>
<tr>
<td><strong>Referral-based — Nonstenotic§</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Grotta et al.</strong></td>
<td>2yr</td>
<td>26</td>
<td>1.9%(5.8%)</td>
</tr>
<tr>
<td><strong>Brewer et al.</strong></td>
<td>1yr</td>
<td>76</td>
<td>0.0%(1.3%)</td>
</tr>
<tr>
<td><strong>Kartchner and McRae</strong></td>
<td>2yr</td>
<td>877</td>
<td>2.0%(1.9%)</td>
</tr>
<tr>
<td><strong>Preoperative§</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Barnes et al.</strong></td>
<td>1yr</td>
<td>35</td>
<td>3% (17%)</td>
</tr>
<tr>
<td><strong>Cooperman and Evans</strong></td>
<td>4½yr</td>
<td>60</td>
<td>3% (4%)</td>
</tr>
</tbody>
</table>

*Incidence rates are approximated from available data for comparison since specific patient-years and mean follow-up are usually not provided.

†Figures include all strokes; if only ipsilateral ischemic stroke is considered, the incidence is nearly the same as in the nonbruit group.

§Long-term incidence, does not include perioperative stroke risk (see table 2).

<table>
<thead>
<tr>
<th></th>
<th>With bruits</th>
<th>No bruits</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Perioperative stroke — Aggregate Data‡</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peripheral vascular surgery</td>
<td>2% (302)</td>
<td>1% (1232)</td>
</tr>
<tr>
<td>Coronary bypass surgery</td>
<td>7% (74)</td>
<td>3% (762)</td>
</tr>
<tr>
<td>General surgery</td>
<td>3% (191)</td>
<td>—</td>
</tr>
<tr>
<td>With stenosis (N)†</td>
<td></td>
<td>No stenosis (N)†</td>
</tr>
<tr>
<td>Peripheral vascular surgery</td>
<td>5% (85)</td>
<td>2.5% (200)</td>
</tr>
<tr>
<td>Coronary bypass surgery</td>
<td>4% (72)</td>
<td>3% (524)</td>
</tr>
</tbody>
</table>

*References 13, 14 and 23 include a small number of patients with prior cerebrovascular symptoms.

†Stenosis implies a 50% or more reduction in lumen diameter estimated by noninvasive techniques; the data of Kartchner and McRae are not included in the table (see text).
Kartchner and McRae reported a 3% stroke rate within two years of follow-up of asymptomatic patients with either abnormal oculoplethysmography or carotid phonoangiography and 12% two-year stroke prevalence when both procedures were abnormal (41% of strokes in this latter group were nonschismic, due to cardiac emboli, or contralateral). Unfortunately, a large percentage of patients were nonrandomly selected for prophylactic endarterectomy, making these results applicable to only a subset: the natural history in consecutive patients could be considerably better or worse. Barnes et al. reported an 8% prevalence of ipsilateral TIA’s (no strokes without TIA) within 11 months of the detection of asymptomatic stenosis discovered in preoperative vascular surgical patients, compared to 2% in such patients without stenosis; they recommended careful follow-up to seek TIA’s rather than prophylactic surgery. As with other series, late deaths due to myocardial infarction were significantly increased with asymptomatic carotid disease.

Asymptomatic Carotid Stenosis Contralateral to Endarterectomy

In an unusual 20-year follow-up series, Levin et al. found that none of 147 patients with asymptomatic carotid stenosis or ulceration contralateral to the endarterectomized and symptomatic carotid artery developed strokes. Seventeen (12%), however, had subsequent TIA’s and were then subjected to endarterectomy. Aggregate data from four studies (table 3) show that stroke incidence is low, with perhaps 10 to 15% experiencing TIA’s which would serve to alert the clinician of impending stroke.

Discussion

The above data briefly summarize the available information on the occurrence and implications of asymptomatic carotid disease. Since asymptomatic bruits are not reliable indicators of carotid occlusive disease, it is not surprising that long-term correlation with ipsilateral ischemic stroke is tenuous. Noninvasive tests by one of several methods are more sensitive and specific. The wide availability of these tests has made the issue of asymptomatic bruit obsolete; rather, the critical issue is of the natural history of various degrees and types of carotid stenosis and ulceration. Further data on this question are urgently awaited.

It is unclear why some patients harbor “benign” atheromas, while in others atheromas are the substrate for stroke. Continued investigation of other risk factors in stroke production (i.e., type and degree of ulceration, local platelet aggregation, cholesterol ester hydrolase activity) may identify atheromas which will benefit from medical or surgical therapy. The use of digital subtraction angiography and real-time Doppler imaging may allow serial assessment of atheromatous ulceration and define their natural history and response to treatment.

Arteriographic investigation and prophylactic carotid endarterectomy are not without definite risk at most institutions; the minimal morbidity reported for these procedures from a few specialized centers cannot be applied to all patient populations, surgeons, and hospitals. Even if successful, carotid endarterectomy does not remove all risk of subsequent ipsilateral ischemic stroke. Justification of prophylactic surgery ultimately requires a randomized trial since the single study purporting to show benefit is obfuscated by nonrandom selection bias.

Conclusions

1. Asymptomatic bruits occur in about 5% of people over age 50 and in 10 to 20% of patients who are about to undergo coronary artery bypass and vascular surgery.

2. Population-based studies show no increase in ipsilateral ischemic stroke in people with asymptomatic bruits.

3. Referral-based studies report a higher than expected stroke rate on follow-up of asymptomatic bruit patients, but methodological flaws limit the possibility of generalization.

4. Asymptomatic bruits detected in preoperative patients undergoing coronary artery bypass or vascular surgery do not reliably predict perioperative, focal stroke risk but may convey increased risk of late stroke and myocardial infarction.

5. The natural history of asymptomatic carotid stenosis detected by noninvasive testing is uncertain. The single consecutive study in preoperative patients shows an increase in late TIA.

6. Contralateral stenosis to an operated artery has a low long-term stroke risk with about 10 to 15% developing TIA’s.

7. There is no convincing evidence that prophylactic carotid endarterectomy is beneficial in stroke prevention in asymptomatic carotid disease.

8. On the basis of the above considerations, we recommend that patients with asymptomatic carotid disease, manifest by bruits or stenosis, be managed by: (a) identification and treatment of cerebrovascular risk factors, especially hypertension; (b) platelet antiaggregation agents (e.g., aspirin 325 mg daily), unless contraindicated; (c) careful follow-up for development of TIA, which would then require further evaluation and therapy; and (d) serial noninvasive testing to detect progressive stenosis, which may require consideration of endarterectomy in selected instances.

Table 3: Asymptomatic Stenosis Contralateral to Endarterectomy

<table>
<thead>
<tr>
<th>Follow-up (yr)</th>
<th>No.</th>
<th>%TIA</th>
<th>%CVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Levin et al.</td>
<td>0–20</td>
<td>147</td>
<td>12%</td>
</tr>
<tr>
<td>Podore et al.</td>
<td>5</td>
<td>25</td>
<td>14%</td>
</tr>
<tr>
<td>Johnson et al.</td>
<td>1–4</td>
<td>22</td>
<td>9%</td>
</tr>
<tr>
<td>Humphries et al.</td>
<td>0–13</td>
<td>168</td>
<td>15%</td>
</tr>
<tr>
<td>Durwood et al.</td>
<td>4</td>
<td>67</td>
<td>13%</td>
</tr>
</tbody>
</table>

*Angiographic stenosis of >50%.
†All patients who developed TIA underwent carotid endarterectomy.
Acknowledgments

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References

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