Ocular Bruits in Ischemic Cerebrovascular Disease

Han-Hwa Hu, MD, Kwong-Kum Liao, MD, Wen-Jang Wong, MD, Michael Mu-Huo Teng, MD, Yuk-Keung Lo, MD, Fu-Li Chu, MD, and Tsuen Chang, MD

A total of 72 ocular bruits in 50 patients with symptoms of atherothrombotic ischemic cerebrovascular disease were studied with continuous-wave Doppler ultrasonography with spectrum analysis (Dopscan). Fourteen patients also had conventional angiography, and 14 had digital subtraction angiography. Ocular bruits by augmentation flow due to occlusion (seven bruits, 9.7%) or tight stenosis (17 bruits, 23.6%) of the contralateral internal carotid artery accounted for only 24 ocular bruits (33.3%). In contrast, siphon stenosis ipsilateral to the ocular bruit was a very common finding. All 14 patients studied with conventional angiography had variable degrees of siphon stenosis ipsilateral to the ocular bruits; there was one angiography failure. We conclude that siphon stenosis can cause ocular bruit alone or can act in combination with augmentation flow by contralateral carotid occlusion or tight stenosis. The difference in their relative occurrence in our patients compared with previous reports probably reflects racial differences in the distribution of stenotic or occluded lesions of the carotid artery between our patients and Caucasian patients. The ocular bruit was the only auscultatory finding in more than a quarter of our patients (14 of 50, 28%). (Stroke 1988;19:1229–1233)

Auscultation of the skull and eyes is now used daily in the practice of neurology. Various conditions such as arteriovenous fistula, cerebral angioma, tumor of the glomus jugulare, angionoma of the scalp, saccular aneurysm, vascular meningioma, compression of a major cerebral artery by a brain tumor, Paget's disease, a highly vascular thyroid gland, coarctation of the aorta, anemia, and even spontaneous physiologic bruits (which are rather common in children), give rise to ocular bruits. Ocular bruits in cerebrovascular disease have been described as unusual. Gautier et al found only one ocular bruit in 150 patients with internal carotid artery (ICA) stenosis or occlusion, and Silverstein et al found none in 30 patients with carotid occlusion. This has led to the widely held opinion that noise heard over the orbit is of little help in establishing the site and severity of lesions of the ICA.

Fisher, however, first reported the occurrence of a cranial (ocular) bruit contralateral to an ICA occlusion and stated that it was a useful additional sign for the clinical diagnosis of carotid artery disease. There is no agreement on the significance of this finding; some texts relate ocular bruits to a stenosis of the intracranial portion of the ICA, but in recent studies unilateral ocular bruits have been interpreted as a sign of augmentation flow due to a tight stenosis or occlusion of the contralateral ICA.

We report a prospective study of 82 ocular bruits heard in 55 patients with symptoms of ischemic cerebrovascular disease and studied with continuous-wave Doppler ultrasonography with spectrum analysis (Dopscan) and/or conventional angiography or digital subtraction angiography (DSA).

Subjects and Methods

From March 1985 to February 1987, we identified 82 ocular bruits in 55 patients admitted to the stroke section with symptoms of atherothrombotic ischemic cerebrovascular disease (stroke or transient ischemic attack [TIA]) and in patients referred to the stroke clinic for evaluation of symptoms suggesting ischemic cerebrovascular disease or asymptomatic carotid bruits.

Auscultation of ocular and cervical bruits was performed with the bell attachment to a standard stethoscope. Three of the authors separately examined all patients for bruits and agreed on the pres-
ence or absence of bruits in all patients. Cervical bruits were classified by location: the common carotid region (base of the neck); the carotid bifurcation region (level of the superior thyroid cartilage and beyond); the angle of the jaw, mastoid region, and supraclavicular fossa. Pitch was judged simply as high or low, and intensity was rated as moderate or loud. Cardiac murmur radiating into the neck was noted especially in reference to any change in pitch and/or intensity at the carotid bifurcation. Auscultation over the orbit was also performed with a bell attachment, the patient either lying or sitting. Patients were asked to close both eyes gently, and the stethoscope was firmly applied over one eye. During auscultation of each eye the patient was instructed to open the other eye to diminish eyelid flutter, which if rhythmic may cause confusion. If a bruit was not readily heard, the patient was asked to hold his breath; if a bruit was heard, both the ipsilateral and the contralateral common carotid arteries were compressed to see if there was any change in the ocular bruit.

All 55 patients had complete physical and neurologic examinations, computed tomography of the brain, blood cell count, T3, and T4 to rule out as many as possible arteriovenous malformation (AVM), tumor, hyperthyroidism, or anemia, which may cause augmentation flow and hence ocular bruits. Five patients were excluded because their ocular bruits might be attributed to anemia (two), carotid-cavernous fistula (two), or AVM (one).

All patients had detailed Dopscan examination using 5-KHz continuous-wave Doppler; after November 1985 duplex scan was also used. The diagnostic criteria for carotid stenosis in our laboratory are Doppler-shifted frequencies of 6–8 KHz indicating 50–75% stenosis, frequencies of 8–12 KHz indicating 75–90% stenosis (50–75% decrease in lumen diameter), and frequencies of >12 KHz indicating >90% stenosis (>75% decrease in diameter). The diagnostic criteria for significant siphon stenosis is no significant stenosis in the ipsilateral extracranial carotid artery and a reversed flow in the ophthalmic artery. The sensitivity and specificity of Dopscan examination in our laboratory has been reported.

We studied 28 patients using angiography; 14 underwent conventional transfemoral angiography with visualization of both extracranial and intracranial carotid arteries, and 14 had DSA (intravenous or aortic injection). The extracranial carotid artery and the ICA siphon were classified as normal, <30% stenosis, 30–50% stenosis, 50–75% stenosis, 75–90% stenosis, >90% stenosis of lumen diameter, or complete occlusion. All angiograms were reviewed by a neuroradiologist who had no knowledge of the presence or absence of ocular bruits or cervical bruits in the ICA.

The ICA was classified as having hemodynamically significant stenosis or tight stenosis when the Dopscan examination revealed a stenotic lesion of >75% decrease in the lumen area, which is an approximately 50% decrease in lumen diameter on the angiogram.

**Results**

We included 43 men and 7 women with 72 ocular bruits in our study. Their ages ranged from 50 to 85 years, with a mean ± SEM of 65.1 ± 7.9 years. Eighteen patients (36%) presented with a stroke; two had reversible ischemic neurologic deficits, 13 had partial nonprogressing strokes, and three had major disabling strokes. Twenty patients (40%) presented with TIAs and 12 (24%) with nonspecific symptoms such as dizzy spells and vertigo. Associated medical conditions were hypertension in 41 (82%), diabetes mellitus in 17 (34%), ischemic heart disease presenting as previous myocardial infarction or coronary artery bypass surgery or history of angina in nine (18%); a cholesterol concentration of >250 mg% and/or a triglyceride concentration of >180 mg% were noted in 16 (32%). Most patients had at least two of the above conditions.

Of the 72 ocular bruits, 28 were unilateral (on the right in six, on the left in 22) and 22 were bilateral. Of the 22 bilateral bruits, 13 were louder on one side than on the other.

Most patients (35, 70%) also had carotid bruits in the neck, and 14 (28%) had vertebral artery distribution bruits in the posterior neck. The ocular bruits were the only auscultatory finding (no neck bruits) in 14 patients, four of whom had unilateral ICA occlusion; nine of the remaining 10 (excluding the one with 20% stenosis of the left ICA) had no demonstrable lesion in the extracranial arteries by Dopscan examination or by angiography (in three). The clinical presentation, ocular bruits, and carotid examination in these 14 patients with only ocular bruits are shown in Table 1. In no patient was the ocular bruit heard ipsilateral to the ICA occlusion. All ocular bruits but one disappeared when the ipsilateral common carotid artery was compressed; in that one patient the bruit increased in intensity when either common carotid artery was compressed and the bruit was thought to arise from the posterior communicating artery. Five patients eventually had a carotid endarterectomy, three for tight stenotic lesions and two for nonsignificant stenoses. The ocular bruit contralateral to the endarterectomy was unchanged in the latter two patients and decreased in intensity in two of the former three patients; it was unchanged in the remaining patient.

Table 2 shows the relation of the 72 ocular bruits in the 50 patients to disease in the ipsilateral and contralateral ICAs, which were classified as completely occluded, tightly stenosed, and normal or hemodynamically nonsignificantly stenosed. The ocular bruits were heard only contralateral to an ICA occlusion but could be heard either ipsilateral (13 of 20, 65%) or contralateral (17 of 20, 85%) to a tight stenosis of the ICA. (Patients with bilateral ocular bruits were counted as having both left and right ocular bruits, and patients with bilateral ICA
TABLE 1. Clinical Presentation and Carotid Artery Features of 14 Patients With Only Ocular Bruits

<table>
<thead>
<tr>
<th>Pt</th>
<th>Clinical presentation</th>
<th>Ocular bruit</th>
<th>Extracranial</th>
<th>Intracranial</th>
<th>Methods</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Left</td>
<td>Right</td>
<td>Carotid artery</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Verteobasilar insufficiency</td>
<td>+</td>
<td>+</td>
<td>Normal</td>
<td>Siphon stenosis, B</td>
</tr>
<tr>
<td>10</td>
<td>Verteobasilar insufficiency</td>
<td>+</td>
<td>-</td>
<td>Normal</td>
<td>1</td>
</tr>
<tr>
<td>12</td>
<td>Infarction, L</td>
<td>+</td>
<td>+</td>
<td>Normal</td>
<td>1</td>
</tr>
<tr>
<td>13</td>
<td>Infarction, L</td>
<td>-</td>
<td>+</td>
<td>Normal</td>
<td>1</td>
</tr>
<tr>
<td>15</td>
<td>Infarction, R</td>
<td>+</td>
<td>-</td>
<td>Normal</td>
<td>1</td>
</tr>
<tr>
<td>16</td>
<td>Dizziness</td>
<td>+</td>
<td>+</td>
<td>Normal</td>
<td>1+3</td>
</tr>
<tr>
<td>31</td>
<td>Infarction, L</td>
<td>+</td>
<td>-</td>
<td>Normal</td>
<td>1+2</td>
</tr>
<tr>
<td>25</td>
<td>Infarction, R</td>
<td>+</td>
<td>-</td>
<td>ICA O, R; normal, L</td>
<td>1+3</td>
</tr>
<tr>
<td>32</td>
<td>Infarction, R</td>
<td>+</td>
<td>-</td>
<td>ICA O, R; normal, L</td>
<td>1+3</td>
</tr>
<tr>
<td>34</td>
<td>Infarction, L</td>
<td>+</td>
<td>-</td>
<td>Normal</td>
<td>1</td>
</tr>
<tr>
<td>35</td>
<td>Dizziness</td>
<td>+</td>
<td>-</td>
<td>ICA O, R; normal, L</td>
<td>1</td>
</tr>
<tr>
<td>37</td>
<td>Infarction, R</td>
<td>+</td>
<td>+</td>
<td>ICA 20% stenosis, L</td>
<td>Siphon stenosis, B</td>
</tr>
<tr>
<td>43</td>
<td>Infarction, L</td>
<td>-</td>
<td>+</td>
<td>Normal</td>
<td>1</td>
</tr>
<tr>
<td>49</td>
<td>Infarction, brainstem</td>
<td>-</td>
<td>+</td>
<td>ICA O, L; 30% stenosis, R</td>
<td>Siphon stenosis, R</td>
</tr>
</tbody>
</table>

Methods: 1, Doppler ultrasonography; 2, conventional angiography; 3, digital subtraction angiography; B, bilateral; L, left; R, right; ICA O, internal carotid artery occlusion.

stenois were also counted as having both left and right ICA stenosis.) Therefore, "augmentation bruits" due to contralateral occlusion (seven, 9.7%) or tight stenosis (17, 23.6%) of the ICA accounted for only 24 (33.3%) of the 72 ocular bruits.

Since it is difficult for DSA and Dopscan examination to demonstrate a lesion in the siphon, the role of siphon stenosis in producing ocular bruits can be discussed only from the findings of conventional angiography. In 14 patients with 21 ocular bruits, conventional angiography always revealed siphon stenosis ipsilateral to the ocular bruits (100%, 20 of 20, one angiography failure) (Table 3). In three patients no ocular bruits were heard ipsilateral to the siphon stenosis; in one (Patient 1), a bruit appeared after walking for 3 minutes and/or after compression of the contralateral common carotid artery, in the second (Patient 33) there was a very tight stenosis in the ipsilateral (right) ICA, and in the third (Patient 31) both anterior cerebral arteries originated from the contralateral (left) ICA. These findings suggest that ocular bruit is strongly associated with siphon stenosis whereas, in contrast, ocular bruits contralateral to complete occlusion or tight stenosis of the ICA were found in only 3 of 21 (14.2%) and 5 of 21 (23.8%) ocular bruits, respectively. Augmentation bruits due to contralateral ICA occlusion or tight stenosis accounted for 8 of 21 ocular bruits (38.0%) in these 14 patients who underwent conventional angiography.

Discussion

Since Fisher’s first report of a cranial (ocular) bruit contralateral to an ICA occlusion,4 Allen and Mustian16 have emphasized the frequent occurrence of ocular bruits contralateral to an ICA occlusion. Bousser et al9 found that ocular bruits are seldom related to carotid siphon stenosis, at least as a single cause; in more than half of their patients there was a tight stenosis (24%, 6 of 25) or occlusion (44%, 11 of 25) of the contralateral ICA with bilateral occlusion in one patient. Pessin et al10 found 76.9% of ocular bruits in their patients contralateral to a tight stenosis (1 of 13) or occlusion (9 of 13) of the ICA.

TABLE 2. Relation of Ocular Bruits to Internal Carotid Artery Disease in All 50 Patients With Atherothrombotic Ischemic Cerebrovascular Disease

<table>
<thead>
<tr>
<th>Ocular bruit</th>
<th>Complete occlusion</th>
<th>Tight stenosis</th>
<th>Normal or nonsignificant stenosis</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>R</td>
<td>L</td>
<td>B</td>
<td>R</td>
</tr>
<tr>
<td>R</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>L</td>
<td>6</td>
<td>0</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>B</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>6</td>
<td>1</td>
<td>0</td>
<td>8</td>
</tr>
</tbody>
</table>

R, right; L, left; B, bilateral. Tight stenosis, >75% decrease in lumen area, ~50% decrease in lumen diameter.
Siphon stenosis, >21% decrease in diameter; R, right; L, left; -, normal; +, <50% stenosis; ++, 50-90% stenosis; ++++, complete occlusion; ( ), blank due to internal carotid artery occlusion.

*Angiography failure.
†Doppler ultrasonographic finding.

In our series only 7 of 72 ocular bruits (9.7%) were associated with contralateral ICA occlusion and 17 (23.6%) were associated with contralateral hemodynamically significant stenosis. In contrast, 100% (20 of 20) of the ocular bruits were associated with ipsilateral siphon stenosis in the 14 patients submitted to conventional angiography, and the nine patients without extracranial artery disease demonstrated by auscultation and Dopscanc examination, or cervical angiography (in three), in whom siphon stenosis was most likely the underlying cause of ocular bruits (Table 1).

We believe that siphon stenosis can cause ocular bruit alone or can act in combination with augmentation flow by contralateral ICA occlusion or tight stenosis. The difference in their relative occurrence in our patients compared with previous reports probably reflects racial differences in the distribution of stenotic or occlusive lesions in our patients and in Caucasian patients. ICA occlusion, either symptomatic or asymptomatic, is often seen in North America and Europe, but is relatively rare in Asia. Differences in the distribution of stenotic or occlusive lesions between Western and Oriental patients was recently stressed in the EC/IC Bypass Study: patients from Europe and North America much more frequently had ICA occlusion and stenosis at the neck, whereas intracranial obstructions were more frequent in patients from Asia.

As Bousser et al stated, ocular bruits in ischemic cerebrovascular disease are heard more frequently than has been assumed. Blood flow in the siphon very easily becomes turbulent and generates ocular bruits. Two possible mechanisms, vortical sounds and turbulence, are germane to arterial murmurs encountered in the cervical and cranial (ocular) regions, and both share the property of being produced by increased velocity and/or decreased viscosity of blood. Vortical sounds result from periodic swirling of blood about a local obstruction in the blood stream. Turbulence refers to completely chaotic blood flow, that is, no well-ordered pattern of velocity of blood in any cross section of the blood stream. Blood flow changes from the usual streamline pattern to turbulent flow when \[ R \sim V D p/\eta \] , where \( R \) is the Reynolds number, \( V \) is the linear velocity of the fluid, \( D \) is the diameter of the tube, \( \rho \) is the density of the fluid, and \( \eta \) is the absolute viscosity, all in metric (centimeter-gram-second) units. In a straight tube, when \( R \) is <2000, flow is believed to be laminar or smooth, but turbulence may appear if \( R \) is >2000 in the case of both water and blood. With deviation of the arterial shape from a straight tube to an S shape, turbulent flow can exist at a lower \( R \). In the carotid siphon, centrifugal thrust of the fast axial current against the wall at a sharp bend results in secondary currents flowing near the walls of the vessel. At the next bend new secondary currents, now in an opposite direction, may induce turbulent flow. This probably accounts for turbulence in the siphon at an \( R \) of only 500, as predicted by Stehbens. Therefore, ocular bruits may easily occur in any situation (such as anemia, contralateral ICA occlusion, siphon stenosis, or a combination of these factors) that increases blood flow or velocity through the ICA and decreases blood viscosity.

There are a number of different mechanisms thought to be implicated in the production of ocular bruits.

1. Increased blood flow velocity through the posterior communicating arteries as suggested by Fisher. This mechanism was thought to be responsible for one ocular bruit in a woman who had tight stenosis of the siphon on the left side and moderate
stenosis of the siphon on the right; the ocular bruit on the left side increased in intensity when either common carotid artery was compressed.

2. Increased blood flow due to both anterior cerebral arteries originating from one carotid artery. We had one patient (Patient 31) whose ocular bruit could be attributed to this mechanism; an ocular bruit was heard on the left side but not on the right despite the fact that the degree of siphon stenosis and condition of the extracranial carotid arteries were very much the same. The only difference was that both anterior cerebral arteries originated from the left ICA, and the ocular bruit may have been heard on the left and not on the right side because of a relative increase of blood flow through the left ICA and a decrease of blood flow through the right ICA.

3. Ocular bruits due to transmission of the louder (contralateral) ocular bruit. This mechanism could explain the situation in which bilateral ocular bruits disappear during compression of one common carotid artery. Twenty-two patients had bilateral ocular bruits, 13 had a bruit louder on one side than on the other. All ocular bruits disappeared only when the ipsilateral common carotid artery was compressed; therefore, no ocular bruit could be attributed to this mechanism in our patients.

4. Compensatory increase in carotid blood flow contralateral to a tight stenosis or an occlusion of the origin of the ICA. This was responsible for 24 bruits, but many were associated with siphon stenosis. Three patients in this group had an endarterectomy ipsilateral to their tight stenosis. After surgery, the contralateral ocular bruit was unchanged in one and the intensity of the bruits decreased in two patients. This, again, suggests that another mechanism, most likely siphon stenosis, also plays a role in the production of ocular bruits.

5. Turbulence and/or an increase in blood flow velocity due to stenosis of the intracrani al portion of the ICA or siphon stenosis. As described, there was a strong association between ocular bruits and siphon stenosis in our patients.

Ocular bruit is a rather common finding in our patients with cerebrovascular disease, and in our study it was the only auscultatory finding in 14 of 50 patients (28%). Four patients with ICA occlusion had only ocular bruits, which means, as in a previous report,¹⁰ that ocular bruits are slightly more common than neck bruits in patients with ICA occlusion (4 of 7, 57.1%).

Ocular bruits occurred only contralateral to an ICA occlusion but could be heard either ipsilateral (65%) or contralateral (85%) to tight stenosis of the ICA (Table 2). Our results reemphasize that ocular bruits, identified by standard auscultation, are strongly associated with siphon stenosis of the ipsilateral ICA and occlusion of the contralateral ICA.

Acknowledgment

The authors wish to acknowledge the generous advice of Professor H.J.M. Barnett.
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doi: 10.1161/01.STR.19.10.1229

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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