Auscultation of Cervical and Ocular Bruits in Extracranial Carotid Occlusive Disease: A Clinical and Angiographic Study

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SUMMARY

The clinical and angiographic features of cervical and ocular bruises were correlated in 50 consecutive patients with severe extracranial internal carotid artery occlusive disease.

Cervical bruises, generally localized to the carotid bifurcation, were highly associated (P = 0.004) with “tight” (residual lumen ≤ 2 mm) internal carotid artery stenosis, but significantly less often with a widely patent or occluded internal carotid artery. Angiographic features of a “slow-flow” state through a patent, but “tight” stenosis were identified as the apparent explanation for the absence of bruist in some patients.

A unilateral ocular bruit contralateral to the side of internal carotid artery occlusion occurred in 9 of 10 patients, more often than an associated cervical bruit, and was interpreted as a sign of augmentation flow.

METHODS

During a 15-month period at New England Medical Center, 50 consecutive patients selected for angiographically demonstrated severe extracranial ICA occlusive disease.
TABLE 1  Angiographic Status of the Extracranial Internal Carotid Arteries (50 Patients)

<table>
<thead>
<tr>
<th>Patient groups</th>
<th>Number of patients</th>
<th>Internal carotid arteries</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>16</td>
<td>tight stenosis &amp; open</td>
</tr>
<tr>
<td>Group 2</td>
<td>11</td>
<td>tight stenosis &amp; occlusion</td>
</tr>
<tr>
<td>Group 3</td>
<td>6</td>
<td>bilateral tight stenoses</td>
</tr>
<tr>
<td>Group 4</td>
<td>16</td>
<td>occlusion &amp; open</td>
</tr>
<tr>
<td>Group 5</td>
<td>1</td>
<td>bilateral occlusions</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
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<tr>
<td></td>
<td>50</td>
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</table>

certical difference in bruit occurrence in “open” or occluded internal carotid arteries.

Five patients had no cervical bruit noted with a “tight” stenosis. In three, the explanation may be found in the angiographic appearance of “slow-flow” through the internal carotid system based on the external carotid circulation filling more quickly than the internal carotid circulation (fig. 1). This angiographic appearance was not present in any other patients. The degree of stenosis in the 3 patients with “slow-flow” was not significantly different from the other patients with bruits. Two patients had no obvious reason for the absence of bruit although both were obese with thick necks, making auscultation difficult.

Bruit occurrence with a “tight” ICA was not significantly different when the contralateral ICA was either “open” or occluded. Bruits were present with “tight” stenosis in 13 of 16 patients (81%) when the contralateral ICA was “open,” and in 10 of 11 patients (91%) when the contralateral ICA was occluded. Bruits were present in association with 11 of 12 (91%) tightly stenotic arteries in the 6 patients with bilateral “tight” ICA stenoses.

External carotid artery anatomy did not account for differences in bruit occurrence in the ICA groups. Ten patients had external carotid stenosis and one patient had an occluded external carotid artery. The external carotid artery stenoses were associated with ipsilateral internal carotid arteries that were either “tight” (N = 5), “open” (N = 3), or occluded (N = 2). The one occluded external carotid artery was associated with an ipsilateral “tight” ICA.

Table 2 presents the findings on cervical bruit location. The majority of bruits associated with “tight” or occluded internal carotid arteries were either localized to the bifurcation region or changed in character (pitch and/or intensity) at that region. By contrast, bruits associated with an “open” ICA were usually heard all along the common carotid artery without change at the bifurcation region.

Cardiac murmurs radiating into the neck were distinguished by auscultation from bruits reflecting carotid bifurcation disease. Six of 7 patients with systolic outflow murmurs had radiation of the murmur into the neck but five of these 6 patient had additional bruits which were loudest at the bifurcation or changed in pitch and/or intensity in this region.

Cervical Bruit and ICA Occlusion

Twenty-eight patients had a total of 29 occluded internal carotid arteries. They were associated with a contralateral “tight” stenosis, “open” carotid, or bilateral occlusions. Seven bruits (24%) were noted in

![FIGURE 1. Left common carotid angiogram, lateral view. There is a ‘tight’ stenosis at the origin of the internal carotid artery. Note that the linear flow in the external carotid system is faster than in the internal carotid system, as evidenced by opacification of superficial temporal artery branches more distally than middle cerebral artery branches. This is a reversal of normal findings.](http://stroke.ahajournals.org/)

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these patients. Five of these bruits occurred in 16 patients with a contralateral “open” ICA, and 2 in 11 patients with a contralateral “tight” ICA. No bruits were present in the 1 patient with bilateral occlusions.

The angiographic configuration of the 29 proximal ICA occlusions, whether a round (N = 13) or pointed (N = 4) stump, or absence of the artery (N = 12), did not account for the occurrence of cervical bruit.

Presumed increased external carotid artery flow, based on retrograde ophthalmic artery filling of the intracranial ICA seen angiographically, was not significantly different in patients with or without a cervical bruit on the side of ICA occlusion.

Cervical Bruit and an “Open” ICA

Cervical bruits were present in association with an “open” ICA in 14 of 32 (44%) patients when the contralateral ICA was “tight” or occluded. There was no significant difference in the occurrence of bruits in an “open” ICA when the contralateral ICA was “tight” (6 of 16) or occluded (8 of 16). There was no apparent difference in the extent of cross hemispheric filling on angiography via the “open” ICA in patients with or without an associated bruit, although filling into the contralateral middle cerebral artery territory was frequently observed in both groups.

Ocular Bruits

Table 3 shows that the majority of ocular bruits (10 of the 13) occurred in patients with ICA occlusion, and were located contralateral to the occlusion in 9 patients. Three patients had ocular bruits with “tight” ICA stenosis, two ipsilateral and one contralateral to the “tight” stenosis.

The ICA on the side of the ocular bruit was “open” in 6 patients and “tight” in 3 patients whose bruit was contralateral to the ICA occlusion. In the remaining 4 patients the ICA ipsilateral to the ocular bruit was “tight” in 2 patients, “open” in one patient, and occluded in one patient. Stenosis of the intracranial portion of the ICA was not a factor in accounting for ocular bruits in any of our patients.

Discussion

The present study focused on selected patients with angiographically proven severe extracranial ICA occlusive disease in an attempt to document the occurrence and clinical and angiographic features of cervical and ocular bruits. We did not study unselected patients with bruits and therefore our findings cannot be generalized to predicting extracranial ICA occlusive disease from clinical bruit analysis alone. Rather, we were interested in establishing that cervical and ocular bruits are a regularly encountered manifestation of severe extracranial ICA occlusive disease.

We found that “tight” ICA stenosis was highly associated with an easily audible cervical bruit. Presumed turbulent flow through the stenotic segment of the artery was sufficient in most cases to produce a bruit, independent of the opposite ICA. Other studies relating cervical bruit to angiographically proven ICA occlusive disease have reported correlations varying from 29% to 88%. A few studies have found occurrence rates comparable to the present report. The variation in the association of bruit with ICA occlusive disease may, in part, reflect the carefulness of auscultation, and the fact that several studies assessed patients with stenoses of only 50%.

A few patients with “tight” ICA stenosis in our study had no audible cervical bruit after careful and repeated examination. A “slow-flow” state through the patent but stenotic internal carotid system was the apparent angiographic explanation in the majority of these patients. Recognition of this “slow-flow” state may be clinically important as a prodromal stage to complete ICA occlusion, while still providing the opportunity for surgical intervention (thrombo-endarterectomy) under appropriate clinical circumstances.

Cervical bruits were associated with ICA occlusion in only 24% of our patients. Other studies have reported the occurrence of cervical bruit with ICA occlusion from 10% to 67%. The mechanism of bruit production in ICA occlusion has been unsettled. Turbulent flow in a proximal, residual stump was not the apparent explanation in our patients since bruits were present in approximately equal frequency whether the angiogram demonstrated a stump (round or pointed) occlusion or complete absence of the ICA. Also, we found no evidence that either ipsilateral external carotid artery stenosis or intracranial collateral circulation from retrograde ophthalmic artery flow were factors in accounting for the bruit heard in association with the ICA occlusion.

The high occurrence (44%) of cervical bruits in association with an “open” ICA may, in part, reflect increased blood flow in the “open” carotid system, since the study’s selection criteria required that the contralateral ICA be severely stenotic or occluded. Some support for this idea derives from the angiographic observations that both cerebral hemispheres received blood via the Circle of Willis from the “open” carotid injection when the contralateral ICA was occluded, and also the fact that cervical bruits, when present, were usually heard all along the carotid artery, not localized to the bifurcation.

The findings of the present study corroborate the earlier observations of Fisher who first reported on the occurrence of a cranial (ocular) bruit contralateral to the side of ICA occlusion. In fact, an ocular bruit was slightly more common than an ipsilateral cervical bruit in our patients. In the majority of patients with an ocular bruit contralateral to ICA occlusion, the carotid system ipsilateral to the bruit was “open,” giving some support to the possibility that increased flow through

<table>
<thead>
<tr>
<th>Table 3 Ocular Bruits</th>
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<tbody>
<tr>
<td>Carotid occlusion</td>
</tr>
<tr>
<td>Contra-lateral</td>
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<tr>
<td>Ocular bruit location</td>
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Effects of Vasoconstriction and Distal Dilation on Carotid Stenoses in the Dog

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SUMMARY Traditionally, arterial stenoses have been assumed to be inflexible, static obstructive lesions that could not acutely change their configuration or cross-sectional area. However, recent clinical and experimental observations have shown that coronary arterial stenoses can respond to vasoconstriction and intraluminal pressure changes. This experimental study evaluated whether similar dynamic changes could occur in a carotid artery stenosis. The effects of dilation distal to a circumferential snare were examined in 6 mongrel dogs. To eliminate collateral flow, the distal carotid artery was occluded and blood flow diverted through a 16 or 20 gauge needle. With no stenosis, dilation caused flow to increase from 29.0 ± 2.0 to 90.1 ± 4.2 ml/min, (p < 0.01). With moderate stenosis, flow increased (25.5 ± 1.3 to 56.4 ± 3.7 ml/min, p < 0.01 following dilation was attenuated. With severe stenosis, flow paradoxically decreased (20.4 ± 1.0 to 11.4 ± 1.0 ml/min, (p < 0.01). This flow decrease was associated with a large stenotic resistance increase (2.13 ± 0.51 to 18.93 ± 5.58 mm Hg/ml.min⁻¹, (p < 0.01). In eight additional experiments, an in vitro preparation was used to examine the effects of vasoconstriction on stenotic severity. Vasoconstriction, induced by ergonovine, methoxamine, angiotensin, or vasopressin, resulted in a significant flow decrease and stenotic resistance increase. Thus, both vasoconstriction and intraluminal pressure were shown to affect stenotic severity, and thereby influence blood flow. These data illustrate hemodynamic factors which may be important in patients with severe carotid artery stenosis.

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TRADITIONALLY, ARTERIAL STENOSES have been assumed to be inflexible, static lesions that could not acutely alter their configuration or cross-sectional area. However, recent clinical and experimental observations of the coronary circulation have questioned this assumption. Coronary arterial stenoses have been shown to respond to vasoconstriction and changes in intraluminal pressure. The effects of dynamic stenoses on the coronary circulation provide a basis for understanding the clinical manifestations of coronary disease, and the observations raise questions concerning similar stenotic effects on the cerebral circulation. This experimental study applied concepts developed from the coronary circulation to evaluate whether similar dynamic changes could occur in extracranial carotid artery stenoses.

Methods

Effects of Distal Dilation

Six mongrel dogs (12.3–16.7 kg) were anesthetized with sodium pentobarbital (30 mg/kg) with supplemental doses as needed. The dogs were given 1,000 units sodium heparin. Via the left femoral artery, a...
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