The Bruit of Carotid Stenosis Versus Radiated Basal Heart Murmurs
Differentiation by Phonoangiography

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SUMMARY Phonoangiography, quantitative analysis of arterial bruits, has been shown to provide accurate noninvasive diagnosis of uncomplicated carotid arterial stenosis, but had not been tested where cervical bruits from other sources were present. In this study, 27 carotid bruits in 15 consecutive patients with carotid bruits and basal heart murmurs were analyzed by phonoangiography. Tape recordings were made over the base of the heart and along the course of the carotid arteries in the neck; spectral analysis was performed as previously described. The spectral shape of the basal heart murmurs was recognizable and amplitude decreased with distance from the heart, although a secondary, lower amplitude, maximum often occurred over the carotid bifurcation. Intrinsic carotid bruits, by contrast, were always maximal over the bifurcation, and although they radiated both proximally and distally, were not detectable over the chest wall. In quantitative terms, the correct diagnosis as to the presence and extent of carotid stenosis was made in 25 of 27 cases (92%) despite the presence of a radiated murmur. Phonoangiography is capable of accurate differentiation of radiated murmurs from intrinsic carotid bruits and of quantitation of the latter even in the presence of radiated sounds.

ATHEROSCLEROTIC STENOSIS OF THE CAROTID BIFURCATION is a well recognized, treatable disease that accounts for 18% of strokes tabulated in the Harvard Cooperative Stroke Registry.¹ It is present in fully 50% of patients with transient cerebral ischemic attacks symptomatically involving the carotid artery territory.² The extent of carotid artery stenosis is usually assessed by X-ray angiography, a technique of established accuracy but with the disadvantage of being an invasive test with varying degrees of morbidity. Phonoangiography⁴⁺ has proved a useful noninvasive method to detect stenosis of the carotid bifurcation when a bruit is present. Unlike many other noninvasive methods for carotid stenosis, it indicates the site and extent of stenosis.³ This technique has been heretofore only for characterizing isolated carotid artery bruits; the phonoangiographic features of cardiac murmurs and of bruits arising from more proximal vessels have not been reported in detail. The present report describes phonoangiographic findings in patients with basal heart murmurs which radiate to the carotid bifurcation and examines differences in those with and without significant stenosis at the carotid artery bifurcation.

Materials and Methods

The 15 consecutively studied patients in this study had bruits arising from the bifurcation of the carotid artery as well as basal systolic murmurs. Each patient was suspected by the referring physician of having carotid stenosis and had a unilateral or bilateral phonoangiogram within 48 hours of a transfemoral carotid arteriogram. Each test was performed and interpreted without knowledge of the results of the other test, i.e., the physician who read the phonoangiogram did not know the results of the arteriogram where the latter was performed first, and vice versa.

Bruitis and murmurs were recorded with the recumbent patient in a quiet room, in a manner described previously.³⁺ The signal from a skin surface piezoelectric displacement transducer (Hewlett-Packard 21050B) was preamplified and stored on magnetic recording tape. Recordings were made over the second right intercostal space, 1 cm below, and 1 cm above the suprasternal notch. The recording microphone was then placed over the right or left common carotid artery, 1–2 cm above the clavicular insertion of the sternocleidomastoid muscle. Further recordings were made at close intervals along the course of the common and internal

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carotid arteries. Generally, this included recording at the level of the lower end of the thyroid cartilage, at the level of the superior aspect of the thyroid cartilage, halfway between the superior aspect of the thyroid cartilage and the angle of the mandible, and at the angle of the mandible.

The recordings were then played into the memory of a Data General Nova 1220 computer via a high speed A/D converter (Analogics AN5800). The digitized signal was displayed on the face of the system's interactive cathode ray tube (CRT) terminal (Tektronix 4010) for analysis. A 50 msec section of the peak systolic bruit from each of 4–6 cardiac cycles was isolated between cursors and returned to the computer for analysis. Likewise, a similar number of segments of diastolic background noise were taken for comparison and subtraction if necessary. The spectrum of each segment was then computed by fast Fourier transform, the results for systole and diastole separately, and the systolic, diastolic, or difference spectrum displayed, as desired, on the CRT. A permanent record was made by taking a Polaroid picture of the CRT face. The semiportable minicomputer system and the entire recording and analysis sequence are described in detail elsewhere.4,5

A murmur or bruit produced by turbulent blood flow has a sound spectrum, when frequency is plotted against intensity, in which the intensity reaches a peak with increasing frequency. As frequency increases beyond the peak, intensity drops smoothly with a characteristic slope.6,4 The frequency peak is called the break frequency (f₀) and is used to determine the residual lumen diameter of the stenotic lesion which produced the bruit.4 For the carotid bifurcation, the simple relationship \( d = \frac{US}{f₀} \) has been shown to be true, where \( d \) is the residual lumen diameter of a stenotic area, \( U \) the linear flow velocity, and \( S \) the Strouhal number, a hydrodynamic constant.7 For the carotid artery, \( U \) can be assumed to be approximately 500, making the calculation of residual lumen size from the break frequency a simple division.4,6

In this study the spectra, recorded at each location on each side of the neck, were compared with respect to the location of the recording, the overall configuration of the spectrum, the locations of the spectral peaks, the intensity of these peaks and the slope of the falloff of intensity after the break frequency peak (fig. 1).

Carotid arteriograms were taken in two planes, corrected for magnification as described previously,4 and the minimal

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**Figure 1.** Frequency-intensity plots (spectral analyses) of recordings of patient 15 (table 2) over the second right intercostal space (2RIS), the suprasternal notch (SSN), the common carotid arteries at the level of the sternocleidomastoid muscle insertion (CCA/SCM), the lower border of the thyroid cartilage (low TC), and upper border of the thyroid cartilage (high TC). The number over each frequency peak represents the intensity relative to the major peak of the basal heart murmur. a) The murmur of aortic stenosis over the 2RIS and the SSN has a break frequency of 70 Hz with small secondary peaks at 110 and 130 Hz. Its radiation is seen in recordings made over the course of the patient's left carotid artery. The frequency peak remains at 70 and 110 Hz in all positions; the intensity over the carotid bifurcation (high TC) is not greater than that recorded over the SSN or 2RIS. Typical spectra of a bruit produced by a tightly stenotic lesion at the carotid bifurcation in the presence of a radiated basal heart murmur are seen in recordings from the same patient's right carotid artery. At the bifurcation, a high intensity 400 Hz peak (consistent with a 1.3 mm residual lumen) is superimposed upon the radiated heart murmur. Recordings at the low TC and CCA/SCM levels show the 400 Hz peak decreasing in intensity with increasing distance from the bifurcation. b) The same patient 1 week after a right carotid endarterectomy. The 400 Hz peak is gone and only the low frequency murmur of aortic stenosis was found over the entire course of both carotid arteries.
TABLE 1. Clinical Characteristics of the Patients Studied

<table>
<thead>
<tr>
<th>Case</th>
<th>Cardiac Diagnosis</th>
<th>Neurologic Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Aortic sclerosis</td>
<td>Transient monocular blindness, right</td>
</tr>
<tr>
<td>2</td>
<td>Aortic sclerosis</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>3</td>
<td>Aortic sclerosis</td>
<td>Transient monocular blindness, right</td>
</tr>
<tr>
<td>4</td>
<td>Aortic sclerosis</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>5</td>
<td>Aortic sclerosis</td>
<td>Left middle cerebral artery embolus</td>
</tr>
<tr>
<td>6</td>
<td>Aortic sclerosis</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>7</td>
<td>Aortic sclerosis</td>
<td>Transient ischemic attack, right</td>
</tr>
<tr>
<td>8</td>
<td>Aortic sclerosis</td>
<td>Transient cerebral ischemia, right</td>
</tr>
<tr>
<td>9</td>
<td>Aortic sclerosis</td>
<td>Left middle cerebral artery embolus</td>
</tr>
<tr>
<td>10</td>
<td>Aortic sclerosis</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>11</td>
<td>Aortic sclerosis</td>
<td>Old left hemisphere stroke</td>
</tr>
<tr>
<td>12</td>
<td>Aortic sclerosis</td>
<td>Right middle cerebral artery stroke</td>
</tr>
<tr>
<td>13</td>
<td>Aortic sclerosis</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>14</td>
<td>Mild aortic stenosis</td>
<td>Transient monocular blindness, right</td>
</tr>
<tr>
<td>15</td>
<td>Aortic stenosis and</td>
<td>Transient monocular blindness, right</td>
</tr>
<tr>
<td></td>
<td>insufficiency</td>
<td></td>
</tr>
</tbody>
</table>

residual lumen diameter measured to the nearest tenth of a millimeter.

Specific catheter-angiographic assessment of the sources of the basal systolic heart murmurs was not made except in one patient. In 13 of the 15 patients, the murmur of aortic sclerosis without a significant gradient across the valve was the clinical diagnosis. In one patient, the diagnosis of calcific aortic stenosis with a modest gradient was supported by phono- and echocardiograms, while in the last patient, rheumatic heart disease with moderate aortic insufficiency and trivial stenosis had been present for many years, had been shown by cardiac catheterization and aortic angiography, and was supported by phono- and echocardiographic data obtained at the time of phonoangiography.

Results

The 15 patients studied had a total of 27 bruits and/or radiated heart murmurs over the carotid bifurcations which were attributed clinically to carotid stenosis (table 1). The 27 carotid bifurcations were divided into 4 groups, based on the angiographic findings (table 2). These groups are 1) open carotid bifurcation with radiated heart murmur; 2) moderate stenosis (2.1 to 3 mm residual lumen diameter) of one of the three vessels at the bifurcation (common, external, and internal carotid), with a superimposed radiated murmur; 3) tight stenotic lesion (0.7 to 1.5 mm residual lumen diameter) of one of the three vessels at the bifurcation, with a radiated heart murmur; 4) occluded internal carotid artery at the bifurcation, with a radiated heart murmur. Five patients underwent endarterectomy and their pathological specimens were further correlated with the phonoangiographic and arteriographic findings.

Table 2. Comparison of Phonoangiographic, Arteriographic, and Pathologic Findings in the 27 Carotid Bifurcations Studied

<table>
<thead>
<tr>
<th>Diagnostic group</th>
<th>Case</th>
<th>Arteriographic residual lumen diameter (mm)</th>
<th>Phonoangiographic diameter (mm)</th>
<th>Lumen diameter (mm) at surgery of int CA</th>
<th>Postop phonoangio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ext CA</td>
<td>Int CA</td>
<td>Ext CA</td>
<td>Int CA</td>
<td>Right</td>
</tr>
<tr>
<td>1</td>
<td>NSS</td>
<td>NSS</td>
<td>NSS</td>
<td>NSS</td>
<td>Rad. bruit</td>
</tr>
<tr>
<td>(no significant</td>
<td>2</td>
<td>NSS</td>
<td>NSS</td>
<td></td>
<td>Rad. bruit</td>
</tr>
<tr>
<td>carotid stenosis)</td>
<td>3</td>
<td>NSS</td>
<td>NSS</td>
<td></td>
<td>Rad. bruit</td>
</tr>
<tr>
<td>4</td>
<td>NSS</td>
<td>2.4</td>
<td></td>
<td></td>
<td>Rad. or 2.5</td>
</tr>
<tr>
<td>5</td>
<td>NSS</td>
<td>2.6</td>
<td></td>
<td></td>
<td>Rad. or 2.5</td>
</tr>
<tr>
<td>6</td>
<td>NSS</td>
<td>2.7</td>
<td></td>
<td></td>
<td>Rad. or NSS</td>
</tr>
<tr>
<td>7</td>
<td>NSS</td>
<td>2.8</td>
<td></td>
<td></td>
<td>Rad. or NSS</td>
</tr>
<tr>
<td>8</td>
<td>NSS</td>
<td>0.7-1.0</td>
<td>1.0</td>
<td></td>
<td>2.5</td>
</tr>
<tr>
<td>9</td>
<td>NSS</td>
<td>1.5</td>
<td></td>
<td></td>
<td>Rad. bruit</td>
</tr>
<tr>
<td>10</td>
<td>NSS</td>
<td>1.2</td>
<td></td>
<td></td>
<td>Rad. bruit</td>
</tr>
<tr>
<td>11</td>
<td>NSS</td>
<td>1.3</td>
<td></td>
<td></td>
<td>Rad. bruit</td>
</tr>
<tr>
<td>12</td>
<td>NSS</td>
<td>0.5</td>
<td></td>
<td></td>
<td>&lt;1</td>
</tr>
<tr>
<td>13</td>
<td>NSS</td>
<td>1.4</td>
<td></td>
<td></td>
<td>1.1</td>
</tr>
<tr>
<td>14</td>
<td>NSS</td>
<td>oocl</td>
<td></td>
<td></td>
<td>Nondefin</td>
</tr>
<tr>
<td>15</td>
<td>NSS</td>
<td>oocl</td>
<td></td>
<td></td>
<td>Nondefin</td>
</tr>
</tbody>
</table>

Abbreviations: Ext and Int CA = external and internal carotid artery; NSS = no significant stenosis, residual lumen diameter ≥2.5 mm; Rad. bruit = radiated bruit; CCA = common carotid artery; Nondefin = nondefinitive; oocl = occluded.

Accuracy of Phonoangiographic Diagnosis

Nine carotid bruits in eight patients were correctly diagnosed by phonoangiography to be radiated heart murmurs and to show no significant narrowing at the carotid bifurca-
tion (table 2, diagnostic group 1). Each of these nine carotid arteries had no significant narrowing on angiography.

Five carotid arteries in five patients were shown on conventional angiography to have moderate stenosis (2.1 mm–3 mm residual lumen diameter) of either the external or internal carotid artery or both, at their origins (table 2, diagnostic group 2). In four of these cases the phonodiographic spectrum was recorded (table 2, diagnostic group 2). In four of these cases the phonodiographic spectrum was read as consistent with moderate carotid stenosis and a residual lumen diameter of greater than 2 mm, with the alternative possibility of a radiated heart murmur. Although the spectra obtained over the carotid bifurcation looked similar to those obtained over the aortic area, they were not identical. When the residual lumen diameter was calculated from their break frequencies, the calculated diameter was in agreement with the arteriogram to within 0.4 mm. The fifth patient (case 8 right) (fig. 2) was diagnosed correctly with regard to the residual lumen diameter, but the bruit was incorrectly attributed to an internal carotid stenosis, while the angiogram showed that only the ostium of the external carotid artery was involved.

Ten patients with 11 tightly stenotic internal carotid artery lesions (1.5 mm or less) near the bifurcation were analyzed (table 2, diagnostic group 3). In every case but one, the bruit arising from the tightly stenotic lesion was correctly distinguished from the superimposed radiated murmur, and a correct lumen diameter (within 0.2 mm) was estimated. The one case that was missed was called a radiated murmur when there was a tightly stenotic lesion, and was therefore considered to be a false negative result. It will be discussed below.

Finally, a radiated murmur was noted in two patients who had an occluded internal carotid artery and an open external carotid artery (table 2, diagnostic group 4). One was correctly diagnosed as a radiated murmur; the other was considered nondefinitive because of a low intensity peak at the bifurcation thought to represent cross neck radiation of a loud carotid bifurcation bruit on the opposite side (fig. 3a). This will be discussed below.

Thus, in the entire group of 27 studies in 15 patients, phonodiagnostic studies gave an accurate diagnosis in 92% of cases (25/27) with one false positive and one nondefinitive spectrum.

Phonodiagnostic Characteristics of Radiated Basal Heart Murmurs Recorded Over a Nonstenotic Carotid Artery (table 2; diagnostic group 1)

Nine carotid arteries in eight patients, which were thought clinically to be stenotic because of well-radiated heart murmurs, were found at arteriography to be patent. In each case, the correct diagnosis was made phonodiagnostically because the overall spectral shape and the break frequency of the sound recorded over the carotid artery along its course were similar to that of the basal murmur. The frequency-intensity characteristics of the murmur of aortic stenosis and their relationship to the gradient across the valve are the subject of another study. The general characteristics, however, are shown in figure 1b for a typical aortic systolic murmur. In most patients a broad peak occurs at between 60 and 100 Hz and a similar one at about 200 Hz. In two of the ten studies, the latter peak was at 280–300 Hz. As one proceeds distally over the common carotid artery away from the second right interspace, the amplitude of the murmur changes but the general shape remains the same. It is often louder over the upper sternum or in the suprasternal notch (fig. 3b). Further away from the sternum along the course of the common carotid artery the amplitude of the peaks generally, but not always, decreases. Sometimes the bruit is more intense over the carotid bifurcations than it is over the common carotid arteries. If that occurs, bruit shape on spectral analysis is similar over both carotids and over the base of the heart, and all sounds may be ascribed to the basal heart murmur.

In two cases there was slight stenosis (residual lumen diameter 3.5 mm) of the internal carotid artery. The

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** The bruit of mild carotid stenosis superimposed on a radiated basal heart murmur (case 8, tables 1 and 2), plotted as in figure 1. The basal heart murmur with a break frequency of 90–100 Hz was loudest over the SSN. A 200 Hz peak, consistent with a residual lumen of 2.5 mm, was seen in recordings made over the right carotid artery and had maximal intensity over the bifurcation (high TC).
Phonoangiographic Characteristics of Bruits in Patients with Tight Carotid Stenosis and a Basal Heart Murmur (table 1, diagnostic group 3)

Eleven bruits in ten patients fell into this category, which included stenotic lesions with residual lumen diameter of 0.7 to 1.5 mm. All of the lesions were at the origin of the internal or at the distal common carotid artery and in each case there was an increase in bruit intensity at the level of the thyroid cartilage or just above it. In all but one case the correct diagnosis of a tightly stenotic lesion at the bifurcation, in addition to a radiated basal heart murmur, was made by phonoangiography. Typical examples of this category are shown in figures 1a and 3a and occurred in nine of the 11 bruits studied. The typical pattern of the basal heart murmur was recorded over the aortic area and low in the neck over the common carotid artery. Closer to the carotid bifurcation a separate higher frequency peak was seen that became more intense over the bifurcation. In case 15, for example, a 400 Hertz peak (fig. 1a) recorded over the tightly stenotic right carotid bifurcation was not seen on the left side, which turned out to be widely patent; there, only the radiated basal murmur of mild aortic stenosis was found. In case 12, a 500 Hz peak recorded over the tightly stenotic left carotid bifurcation was also present at much lower intensity over the right carotid bifurcation. This was thought to represent radiation across the neck of the left carotid bifurcation bruit (see below).

Five of the 11 patients had endarterectomies; in each case, the bruit with the high break frequency was no longer

Phonoangiographic Characteristics of Bruits in Patients with Mild Carotid Stenosis and a Basal Heart Murmur (table 2, diagnostic group 2)

Five carotid arteries in five patients were demonstrated at arteriography to have moderate carotid bifurcation stenosis with residual lumen diameters of 2.1 to 3 mm (40 to 60% stenosis). Phonoangiographic analysis of four of the five bruits showed that they had a similar shape and configuration to the basal heart murmur heard lower in the neck and over the chest. In each case the intensity of the murmur at the bifurcation was less than its intensity lower in the neck or over the chest. In these four cases the moderate stenosis was apparently not producing a bruit at all or not producing one loud enough to be appreciated as separate from the radiated basal murmur.

In the fifth patient, (fig. 2) a separate 200 Hertz peak, not present in the basal heart murmur, was recorded over the common carotid artery and, at somewhat greater intensity, over the bifurcation. We concluded that the sound recorded over the bifurcation in this case was the summation of a bruit produced by a mildly stenotic lesion and a basal heart murmur, the former radiated faintly down the course of the common carotid artery and the latter radiating upward.
recorded postoperatively. Instead, the phonoangiogram over the operated bifurcation took on the configuration of the radiated basal heart murmur (figs. 1b and 3b). The pathological specimen in each of these five cases was cut in cross section; the residual lumen diameter in each instance correlated to within 0.5 mm with the phonoangiographic calculated residual lumen diameter (table 1).

The one false negative result, case 4, had a basal murmur with a high break frequency of 300 Hz over the aortic area and common carotid artery. A second peak of low intensity at 450 Hz was present over the bifurcation giving a "double hump" configuration to the phonoangiogram at this location. The phonoangiogram was incorrectly interpreted as indicative only of the radiated heart murmur, even though the second, high-frequency peak was not seen on the opposite side of the neck. Had the second peak been called significant, the calculated residual lumen diameter would have been 1.1 mm. The angiographic diameter was 1.5 mm. A subsequent patient (case 14) had a basal heart murmur with a break frequency of 300 Hz and a separate bruit coming from a tightly stenotic carotid lesion. The higher frequency peak (450-600 Hz) was maximal over the right carotid bifurcation while the lower frequency peak of 300 Hz decreased in intensity as one ascended from the base of the neck toward the carotid bifurcation. The bruit over the opposite bifurcation, which was angiographically "open," had only a 300 Hertz peak. In this case the correct diagnosis was made by phonoangiographically following the radiated murmur up both sides of the neck and noting the extra peak at 450-600 Hz over the right as compared with the left carotid bifurcation.

Phonoangiographic Characteristics of Bruits in Patients With an Occluded Internal Carotid Artery and a Basal Heart Murmur (table 2, diagnostic group 4)

Two cases fall into this category. The phonoangiogram of one of these (case 11, table 1) appeared similar in configuration along the course of the left common carotid artery, over the bifurcation, and over the base of the heart. The bruit over the bifurcation was therefore diagnosed as indicative of a radiated basal heart murmur. In this instance the occluded internal carotid artery did not give rise directly or indirectly to any detectable sound separate from the radiated basal murmur. The second patient (fig. 3a) had a phonoangiogram that showed a very low intensity 500 Hz peak over the right carotid bifurcation that was not seen on analysis of the basal heart murmur or over the right common carotid artery. The phonoangiogram was read as nondefinitive, although the interpreter suggested that the very low intensity 500 Hz peak was the result of cross-neck radiation of the loud bruit arising from the patient's tightly stenotic left internal carotid artery. In support of this conclusion the 500 Hz peak was no longer present on either side after a left internal carotid endarterectomy (fig. 3b).

Discussion

Phonoangiography is based upon the hypothesis that bruits arising from stenotic arteries are due to turbulent flow and that the sound spectrum of the bruit is quantitatively related to the diameter of the residual lumen at the stenosis. This theory was proved valid in laboratory studies of dog carotid and femoral arteries with experimental stenosis and in human subjects with carotid stenosis. The typical and usual configuration of the frequency-intensity spectrum of a bruit resulting from turbulent flow is relatively flat or rises to a discrete peak, the break frequency, beyond which it falls with a characteristic slope. Less often however, this typical spectrum may not be found and a single break frequency can not be obtained. A series of peaks may appear, without a single point where a dramatic decrease in intensity can be seen that could be called a clear break frequency. Alternatively, bruit amplitude may be so low that, although it can be heard with the ear, it cannot be distinguished in the computer from background noise. Lastly, a broad frequency-intensity plateau may appear with no clearcut break frequency.

This study was undertaken because 16 of 60 carotid bifurcation bruits in an earlier series were uninterpretable for one of the above reasons. Four of these bruits arose from significant carotid stenotic lesions. Review of the uninterpretable bruits from that study suggested that the bruit recorded over the carotid bifurcation was in some cases the sum of two sounds — the intrinsic carotid bifurcation bruit and a bruit radiated from the heart or the great vessels. Accordingly, our technique was changed and recordings were routinely made and analyzed at several locations between the base of the heart and the base of the skull.

Although there are many potential sources of radiated bruits heard over the carotid bifurcation, all of our patients were suspected of having murmurs arising from the aortic valve. In two of our patients aortic stenosis of mild to moderate degree (in one case accompanied by aortic regurgitation) was clearly present. In the remaining 13 patients a basal systolic murmur was present with no evidence of obstruction; in all of these 13 patients, the clinical diagnosis of aortic sclerosis was made. Potentially, rapid flow in a widely patent vessel may become turbulent and widespread murmurs and bruits may be heard in patients with very high blood flow, as in severe anemia. None of our patients was in a high-flow state.

The murmur or bruit was always maximal over the site of its production and generally decreased in amplitude as one listened or recorded proximally and distally. In some instances, however, a radiated heart murmur was louder near the carotid bifurcation than lower in the neck, presumably because the carotid bifurcation is closer to the skin than are the common carotid and subclavian arteries. Because the sound spectra of intrinsic carotid bruits usually differ from those of heart murmurs — exhibiting higher break frequencies and much less energy at very low frequencies — misinterpretation of the louder bruit at the bifurcation ordinarily does not occur. Sometimes, however, it may not be possible to distinguish an intrinsic carotid bruit with a low break frequency from a radiated basal heart murmur. Since a low break frequency (150-250 Hz) signifies no hemodynamically significant stenosis, such a coincidental similarity between heart murmur and carotid bruit does not give a false negative result, i.e., failure to diagnose significant carotid stenosis. Similarly, when the basal heart murmur has a high break frequency (e.g. 300 Hz), it may be close to that of the carotid bruit. The latter may not be seen, or may appear as a second, slightly higher frequency peak. Often, comparison of the spectra recorded over both carotid bifur-
cations may help to demonstrate the presence of a unilateral intrinsic carotid bruit in these two situations. One other potential source of error is that as the arterial stenosis increases toward total occlusion, the intensity of the bruit decreases. It may become of lower intensity than the radiated murmur, or so faint that its signal-to-noise ratio is too low for spectral analysis. In our experience, this occurs very rarely.

In summary, recording of murmurs and bruits over the base of the heart and along the course of the carotid arteries, combined with spectral analysis, usually permits differentiation of intrinsic carotid artery bruits from those due to radiated basal heart murmurs and allows quantitation of the extent of carotid stenosis even in the presence of radiated murmurs.

References


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Long-term Mitral Valve Replacement in Young Children

Influence of Somatic Growth on Prosthetic Valve Adequacy

SIDDNEY FRIEDMAN, M.D., L. HENRY EDMUNDS, JR., M.D., AND CHARLES C. CUASO, M.D.

SUMMARY Long-term clinical and laboratory findings in three children who required mitral valve replacement below age four years are reported. In each instance a second valve replacement was necessary approximately 8½ years after the initial one, following a two and one-half fold increase in body weight. Inadequate mitral valve orifice size was found in each instance, producing a hemodynamic picture equivalent to mitral stenosis: congestive heart failure, pulmonary hypertension and atrial fibrillation. A second valve was placed without mortality in each instance and relieved the mitral valve obstruction. Pulmonary vascular resistance increased postoperatively in two patients and failed to decrease in the third. Pulmonary arterial hypertension and left ventricular hypertrophy persisted as long as 13 to 37 months after the second valve placement in all patients. The consequences of increasing body size and the long-term interposition of a rigid prosthesis in a growing heart introduce additional complications to mitral valve replacement in childhood. Frequent hemodynamic observations and the use of a prosthesis other than the ball-cage variety is recommended for improved management.

SEVERE MITRAL VALVE DEFORMITY producing intractable heart failure is an indication for prosthetic valve replacement even in very young patients. The clinical and hemodynamic improvements which follow valve replacement are often dramatic and the long-term hazards of this procedure have been steadily decreasing as improved valve forms and surfaces have been developed.1-3 Well recognized complications of prosthetic valve use are thromboembolism, bleeding related to anticoagulation, bacterial endocarditis, cardiac arrhythmias and prosthesis breakdown; these occur with approximately equal frequency in adults and children.4 In the pediatric age group, an additional consideration is the effect of body growth on the long-term functional adequacy of a prosthetic device, and the in situ effect of the valve prosthesis on growing myocardium.

The purpose of this report is to describe the clinical, hemodynamic and surgical observations in three children who were treated by mitral valve (Starr-Edwards) replacement under the age of four years and who were subsequently followed for periods of 7½, 9 and 9½ years prior to reoperation. During these intervals, each child showed a near normal somatic growth rate and was relatively free of cardiac symptoms. Eventually, however, all three developed serious cardiac problems which necessitated a second, larger mitral valve prosthesis.
The bruit of carotid stenosis versus radiated basal heart murmurs. Differentiation by phonoangiography.
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