effects of spontaneous clearing of a dissection, the fragmenting of an embolus, the resolution of spasm and the results of surgical by-pass procedures on flow. Such quantitative information might help us to understand better the mechanisms of ischemia in the presence of carotid lesions.

References

SUMMARY One hundred and sixteen carotid artery bruits were assessed using quantitative phonoangiography (spectral bruit analysis — SBA). This technique uses the averaged break frequency of the bruit to calculate the diameter of the residual lumen at the site of stenosis. Biplanar contrast arteriography was performed on 43 (37%) of the sides. All 116 sides were also evaluated with an ultrasonic Duplex scanner. Ten (8.6%) bruits could not be analyzed by the SBA, leaving 106 sides in which the residual lumen diameter could be estimated. The diameter of the vessel at the site of stenosis estimated by SBA and arteriography were compared and found to agree within 1 mm of each other in 85% of patients. A linear relationship was demonstrated between absolute lumen diameter and percent stenosis as measured from the arteriograms, but we were unable to correlate the absolute diameter of the residual lumen as assessed by arteriography or SBA with the assessment of the degree of the stenosis derived from spectral analysis of the pulsed Doppler signal. The significance of these findings is discussed with relevance to the clinical application of spectral bruit analysis.

Quantitative Carotid Phonoangiography

Methods
The patients in this study were all consecutive referrals to the non-invasive laboratory for ultrasonic Duplex scanning of their extracranial arterial system. They all had neck examination with a stethoscope and if a bruit was present, a quantitative phonoangiogram with spectral bruit analysis (SBA) was performed. All patients also had a pulsed Doppler assessment of both carotid systems, but the decision to obtain an arteriogram was made at the discretion of the referring physician.

Those who had arteriography had planar views of both carotid bifurcations. The films were read by a radiologist who was unaware of the test results. The diameter of the lumen at the site of the stenosis was measured directly from the arteriogram using calipers and corrected for the effect of magnification. The degree of stenosis of the internal carotid artery was...
also classified by the percentage reduction in the diameter of the vessel.

The bruits were recorded with the patient supine, in a quiet room using a Hewlett Packard piezoelectric displacement microphone (Model 21050 A/B). Recordings were made along the course of the common carotid artery from the level of the clavicle cephalad to the angle of the mandible in order to determine the region of maximal intensity of the bruit.

The bruits were analyzed by a microprocessor to produce a mean intensity spectrum (fig. 1). The bruit is recorded and displayed on the cathode ray tube to allow identification of systole and diastole. A 50 m/sec "window," appearing as vertical cursors on the screen, is manually positioned "on the bruit" at the location where the analysis is to be performed. This position should include the point of maximal intensity of the bruit which may be identified on the single stored cardiac cycle displayed on the screen. The computer then performs a fast Fourier transform on the spectra from 6 cardiac cycles over this 50 m/sec window. This result is averaged to produce the mean intensity spectrum. The intensity spectrum had to meet the characteristics of turbulence as defined by Fredberg; otherwise the spectrum is unsuitable for analysis. These characteristics of turbulent flow within the vessel are such that the intensity of the sound produced declines as frequency increases beyond a single identifiable frequency (the break frequency) (fig. 2).

Figure 1. The mean intensity spectrum of a single bruit with the cursors positioned, as displayed on the cathode ray tube (CRT).

Figure 2. The break frequency of the bruit indicated by the vertical line as displayed on the CRT.
The diameter of the residual lumen was calculated from the equation $D = \frac{u}{f_0}$, where $u$ is the peak systolic velocity in the unoccluded portion of the artery and $f_0$ the break frequency derived from the spectral analysis. The peak systolic velocity, $u$, is assumed to be 500 mm/sec as derived by Barrett in a series of experiments on the human.

The pulsed Doppler recordings are made using a Duplex scanner combining B-mode and 5 MHz pulsed Doppler, thus allowing accurate placement of the sample volume at any point within the desired vessel. The B-mode image was used merely as a guide to the placement of the sample volume, no measurements of the diameter of the vessel were made from it. The velocity waveform is analyzed by a fast Fourier transform spectral analyzer to produce a hard copy output of the frequency content of the back scattered Doppler signal. This information is used to classify the degree of stenosis of the vessels into 5 groups: A (normals), B (< 10% stenosis), C (10-49% stenosis), D (50-99% stenosis) and E (total occlusion) by criteria previously published from this laboratory. It is not possible at present to make finer gradations of the degree of the stenosis from the data derived from the Duplex scanner.

**Results**

Five hundred and seventy-five patients were referred to our laboratory during the study period. One hundred three of them had bruits over the carotid bifurcation. Ninety patients had unilateral and 13 bilateral bruits, making 116 bruits available for study. Forty-three arteries from which bruits were heard had arteriography and were available for comparison of SBA with arteriography (table 1).

Ten patients with unilateral bruits could not be evaluated. Seven bruits could be heard with the stethoscope but were not heard using the microphone of the spectral bruit analyzer. In the remaining 3 patients no definite break frequency could be identified, hence, these bruits could not be analyzed.

There was agreement within 1 mm between SBA and arteriography in 85% of the bruits that could be analyzed. Agreement was found in 87% of cases if the limit was increased to 1.5 mm. In 4 sides the diameter was overestimated by greater than 1.5 mm using SBA (table 2). One side with an occlusion of the internal carotid had a bruit with a break frequency that gave an estimated residual diameter of 1.4 mm. The relationship between the diameter of the vessel estimated by SBA and arteriography is shown in fig. 3, and is seen to be linear ($r = 0.67, p = 0.00002$). There is also a linear relationship between the percent stenosis of an internal carotid artery and its residual diameter ($r = 0.76, p = 0.0001$) (fig. 4) as calculated and measured from the arteriograms in this series.

Sixty-six internal carotid arteries were assessed by Duplex scanning and spectral analysis as having a greater than 50% diameter reduction (D). Twenty-three internal carotid arteries were estimated by Duplex scan and spectral analysis to have a 10-49% stenosis (C) and 15 less than 10% stenosis (B) (table 3). Two internal carotid arteries were found to be occluded. Thirty of those in group D, 9 in group C and 2 in group B had arteriography. Within these groups, arteriography and SBA agreed within 1.0 mm of each other in 80%, 86%, and 0% of cases respectively (table 3). However, the scattergram in figure 5 shows that there is a wide scatter in size within each Doppler group. There was no significant difference in the diameter of the vessels as estimated by SBA and arteriography between the Doppler groups B and C, C and D, or B and D ($p = 0.06$ Mann-Whitney U Test).

**Discussion**

Estimates of the incidence of the association of bruits and extracranial carotid disease vary. Gilroy and Meyer in a study of 50 symptomatic patients with

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**Table 1** Number of Patients and Sides Examined

<table>
<thead>
<tr>
<th>Number of patients referred</th>
<th>575</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients with bruits</td>
<td>103</td>
</tr>
<tr>
<td>Number of bruits</td>
<td>116</td>
</tr>
<tr>
<td>Number of bruits able to be analyzed</td>
<td>106</td>
</tr>
<tr>
<td>Number examined by SBA and arteriography</td>
<td>43</td>
</tr>
</tbody>
</table>

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**Table 2** Limits of Size Showing Number and % Agreement Within These Limits

<table>
<thead>
<tr>
<th>Limit of Size</th>
<th>Number Agreeing Within Limits</th>
<th>% Agreement Between SBA and arteriogram</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5 mm</td>
<td>31</td>
<td>75</td>
</tr>
<tr>
<td>1.0 mm</td>
<td>35</td>
<td>85</td>
</tr>
<tr>
<td>1.5 mm</td>
<td>36</td>
<td>87</td>
</tr>
<tr>
<td>1.5 mm 3.5</td>
<td>40</td>
<td>97</td>
</tr>
</tbody>
</table>

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**Figure 3.** Scatter diagram of the estimate of the residual lumen by the phonangiogram against the measured diameter from the arteriogram for 41 sides. ($r = 0.67, p = 0.00002$). Two sides had arteriography and were unable to be analyzed by the SBA. One of these was occluded.
arteriographic evidence of disease found a bruit in 57%, this being in agreement with the findings of Peart and Rob. Silverstein and his coworkers heard a carotid bruit in only 13% of their patients with arteriographically proven carotid stenosis. These figures are at variance with those of other authors. Shapiro and co-workers found a correlation between the presence of a bruit and disease in 71.5% of their patients. However, all of their patients had a 50% or more stenosis of the underlying vessel. David and co-workers noted that in a group of patients with transient ischemic attacks, 75% of the carotid bruits were associated with stenotic lesions. In asymptomatic patients, 65% of the vessels with bruits were associated with disease. In a study of a group of patients with cerebrovascular insufficiency undergoing physical and arteriographic examination, Ziegler and co-workers failed to demonstrate a bruit associated with stenosis of the internal carotid artery in 73% of the 199 patients studied. They also noted a bruit in 10% of patients in whom there was no underlying carotid disease. In the series reported here 18% of the patients had a carotid bruit.

A bruit may arise from a stenosis in the external carotid artery and be thought to originate from the internal carotid artery. This might explain the two outlying values indicated on figure 3, although the estimated diameter in these patients appears to be rather large for external carotid arteries (3.1 mm and 3.6 mm by SBA). No maneuvers were performed to confirm whether the external carotid artery was the site of origin of these bruits, but in these patients the external carotid artery had a 40% stenosis on arteriography. None of the other internal carotid arteries having arteriography in this series was associated with external carotid stenoses greater than 20%.

The presence of bruits over the carotid artery is therefore a fallible indicator of internal carotid artery disease. Large numbers of stenotic lesions will not be...
detected by auscultation of the neck and, as such, the presence of a bruit should not be used as the sole criterion for the diagnosis of disease. This imposes severe limitations on the application of SBA.

Ten bruits in our series (8.6%) were unsuitable for analysis, either because these were not heard using the microphone (7/10) or because a definite break frequency could not be identified (3/10). Duncan and coworkers found that ten spectra from a total of 50 which they examined in their series were unsuitable for analysis. These represent a significant proportion of an already small group of patients.

There is evidence that the degree of narrowing of a vessel seems to have some influence on the production of a bruit. In the present series, of those vessels assessed by arteriography, 30 bruits were detected in vessels whose arteriographic diameter was less than or equal to 2.0 mm whereas only 11 vessels greater than 2.0 mm in diameter had bruits originating from them. This would seem to lend support to the contention that greater degrees of stenosis are more likely to produce bruits. Further support for this is seen in the finding that the majority of the sides over which a bruit was heard had stenoses greater than 50% by the pulsed Doppler criteria (n = 66). Spectral bruit analysis relies on the theory that the tighter the stenosis the higher the break frequency, i.e., the higher the frequency at which the intensity will first begin to fall, therefore permitting identification of the break frequency. This situation may be expected to occur in an occlusion of the internal carotid artery when increased flow in the contralateral internal carotid may result in a bruit on that side. Ziegler et al. found this situation in 3 of their patients, no disease being demonstrated on the side of the bruit. Such a situation may lend the observer to the erroneous conclusion that there is disease on this side.

A great deal of experimental work has been devoted to the definition of a critical stenosis, which may be defined as that degree of narrowing at which a measurable pressure drop occurs across the stenosis. Brice demonstrated that despite the original area of the internal carotid artery, it was not until a crosssectional area of 5 sq mm was reached that "significant" decrease in flow and pressure occurred. DeWeese and associates, using pressure measurements in carotid arteries of 61 patients, demonstrated that stenoses of greater than 47% diameter reduction caused drops in mean pressure greater than 10 mm Hg. These studies tell us little about the effects of the stenosis on the higher harmonics of the pulse pressure wave which could be of use in estimating lesser degrees of stenosis. Spectral analysis of back-reflected Doppler ultrasound can be used to detect flow reducing lesions and is sensitive to lesser degrees of stenosis.

The 5 degrees of stenosis used in the spectral Doppler grading of internal carotid artery disease have been shown to correlate well with the arteriographic percentage stenosis of the vessel. However, we were unable to correlate the absolute vessel diameter with the Doppler grading of the lesion in this study. A decrease in absolute vessel diameter, as measured by both arteriography and SBA, may be seen between the 10-49% stenosis and the > 50% stenosis groups in figure 4, but this difference was not statistically significant. This trend may indicate that the character of the plaque itself will influence the grading of the lesion using velocity patterns. Rough, ulcerated lesions could be expected to produce more disturbance of flow than smooth lesions resulting in increased broadening of the frequency spectrum of the velocity wave and, therefore, may be graded as more severely diseased than their residual lumen diameter would indicate.

A further reason for this apparent anomaly may result from the fact that the Doppler data are used to categorize disease into broad groups. Within each of these groups there may be a wide variation in the diameter of the residual lumen. This will be detected by SBA, but, as yet, the methods of analysis of the spectrum derived from the Doppler shift do not permit such a fine gradation.

Spectral bruit analysis can, therefore, be used to assess the residual lumen diameter in internal carotid arteries, but bruits arising in the external carotid are a potential source of misinterpretation. The residual lumen diameter allows one to estimate the percent stenosis of the internal carotid artery. Unfortunately, the technique is only applicable to that small percentage of cases with a bruit; but it may be of use in further defining the size of a vessel which has been assessed by other non-invasive techniques. In this respect it should prove a valuable tool in following patients with asymptomatic bruits and may detect disease progression in these patients much earlier than conventional non-invasive means.

References

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EXTRA-INTRACRANIAL microanastomosis between the superficial temporal artery (STA) and cortical branches of the middle cerebral artery (MCA) has been performed by many neurosurgeons. The bypass procedure has been applied to the treatment of patients with extra- or intracranial occlusive arterial lesions,\(^4\) and to the prevention of stroke resulting from surgical occlusion of cerebral arteries in patients with giant aneurysms or tumors.\(^5\) The technical success of the STA-MCA anastomosis has been evaluated chiefly by clinical symptoms, cerebral angiography,\(^6\) and cerebral blood flow studies using radioactive tracers.\(^7\) Cerebral angiography is traumatic and difficult to perform repeatedly. Cerebral blood flow studies are quantitative, but extracranial contamination is inevitable.

Ultrasonic Doppler flowmetry has been gaining acceptance as a non-invasive method for determining the patency of the STA-MCA anastomosis.\(^8\) The accuracy of Doppler sonography has already been investigated by comparison with angiography\(^9\) and the bypass patency checked by detecting the change in the Doppler flow pattern of the STA.\(^10\) In these studies, the objective was to determine by a qualitative evaluation whether the bypass was patent. If a quantitative value could be attained by Doppler technique, the findings might give information useful in selection of candidates for a bypass and assist in long term follow up.

This study evaluated the effect of the shunt operation quantitatively using the Doppler flowmetry and the findings were compared with the results achieved by cerebral angiography.

**Clinical Materials and Methods**

Ultrasonic Doppler studies were performed pre- and postoperatively in 22 patients who had STA-MCA anastomosis. Twenty-four STA-MCA anastomoses were performed in these patients (2 patients having bilateral procedures). The age of the patients ranged from 38 to 65 with a mean of 52.0 years; 2 patients were females, 20 males.

Table 1 shows the preoperative findings in the patients. Clinical manifestations were transient ischemic attacks (11 patients), mild to moderate neurological deficit (10 patients), and eye-lid ptosis and double vi-
Quantitative carotid phonoangiography.
R Knox, P Breslau and D E Strandness, Jr

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