Phonoangiography by Autocorrelation

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SUMMARY Phonoangiography, as a noninvasive quantitative analysis of arterial bruits, was conducted just prior to standard radiographic angiography in 135 patients. Sound records from 162 carotid arteries were analyzed with a new processing technique employing a high speed analog acoustic analyzer, the autocorrelator. In 18 arteries with carotid stenosis, a correlation coefficient of 0.87 resulted between phonoangiographic diameter predictions and radiographic diameter estimates. Bruit analysis identified two patients with patent lumen diameters, but tortuous carotid arteries. One hundred thirty-three carotid arteries had no bruits that could be analyzed, but angiograms showed no extracranial stenosis. Four arteries from which bruits could not be analyzed were found to be totally occluded. Carotid phonoangiography appears applicable in approximately one of seven patients now requiring angiography of head and neck vessels. When applicable, phonoangiography is significantly correlated with radiographic findings.

IT IS DESIRABLE to supplement arteriographic diagnosis of vascular disorders with reliable noninvasive techniques. Phonoangiography, described by Lees and co-workers, is a procedure for quantitative analysis of arterial bruits. Analysis of 50 carotid bruits in 48 patients by these investigators provided estimates of residual carotid lumen diameters which differed from the radiographic values by less than 1 mm in 83% of cases studied. The patients reported by Lees were selected because of identified cervical bruits.

We report here a second evaluation of the method in a patient group selected because of a need for cerebral or cervical angiography. We have employed a different procedure for bruit analysis. Our findings confirm the validity of the original hypothesis underlying phonoangiography and extend previous observations regarding a problem with the method when stenosis severely reduces flow. In addition, we have found that two bruits associated with tortuous carotid arteries, but patent lumen, could be differentiated from major stenosis by this method.

Methods

Patients

During a period of four months, patients over 40 years of age undergoing carotid and/or aortic arch arteriography at Los Angeles County — USC Medical Center were sound-recorded and evaluated for turbulent bruits. In addition, 51 patients under 40 years whose clinical histories suggested vascular disease and 36 patients with trauma involving the arteries of the neck were recorded.

Procedures

A miniature Sony electret condenser microphone model ECM-50 was adapted for skin application with a parabolically shaped polycarbonate bell. Acoustic signals were selectively amplified to enhance turbulent spectral characteristics. A variable-bandwidth Princeton Applied Research model 113 preamplifier was set for low-frequency roll-off at 1000 Hz, a high-frequency roll-off at 3000 Hz and a gain setting of 500. Signals were stored with a Tandberg Cross-Field Series 3300X tape recorder.

Transmitted heart sounds and background noise were defined initially by inching the microphone from the base of the neck to the angle of the mandible and accepting for analysis only those individuals whose bruits were loudest near the carotid bifurcation. In subsequent batch processing of recorded data, bruits were rescanned for predominantly turbulent flow sounds on an oscilloscope display. Figure 1 illustrates an acceptable bruit sequence.

Criteria for acceptability were low background noise and the abrupt onset and gradual decay of sound, each sound burst occurring periodically with each pulse wave. Recordings of bruits were discarded when the bruit signal was low in relation to baseline noise or if the only significant signal was the transmitted pulse waves or heart sounds (fig. 2). A
turbulent time scale, $\tau_0$, was next obtained from the width of the autocorrelation function.* $\tau_0$ is a reciprocal of the peak frequency, $f_0 = 1/2\tau_0$, which is closely analogous to the "break frequency" obtained from power spectral analysis by Lees and co-workers.† Residual lumen diameter of vessels was estimated using the Strouhal relationship, $fd/U = 1$, or $d = U/f_0$, where $d$ equals the residual diameter, $U$, the peak systolic flow velocity in the unoccluded artery distal to the stenosis and $f_0$ is the unique peak frequency. Peak systolic flow velocity, $U$, was assumed to be 50 cm/sec.† A detailed discussion of bruit screening and analysis by autocorrelation is presented in an Appendix.


†We wish to acknowledge the generosity of R. S. Lees and C. F. Dewey, Jr. in providing tape recorded bruits and analytic data from power spectral analysis.

Figure 1. Two-second oscilloscope sequence showing two acceptable bruits and the onset of a third; criteria for acceptability were the obvious onset and gradual decay of random turbulence, each turbulent burst occurring periodically with each pulse wave, and low background noise with respect to the turbulent signal.

Figure 2. Two-second oscilloscope sequence illustrating unacceptable data; no obvious turbulent bruit is evident over background noise level; the only signal of significance is the transmitted heart sound.

Figure 3 illustrates results when eight carotid bruits previously recorded and analyzed by Lees and co-workers† were analyzed by autocorrelation. The correlation coefficient between break frequencies calculated from Fourier analysis and autocorrelation was $r = 0.98$.

Results

One hundred sixty-two arteries in 135 patients were recorded over the region of both carotid bifurcations. The results are shown in table 1. Phonoangiography was an effective predictor of carotid stenosis for 18 arteries in 14 in-
individuals. A correlation coefficient, \( r = 0.87 \), was found between phonoangiographic diameter predictions for maximal carotid stenosis and values independently estimated from carotid radiograms (fig. 4). In these patients, phonoangiography could have reduced the need for radiography.

Recordings of four arteries in four individuals contained analyzable turbulent bruits but no carotid narrowing was evident in their arteriograms. The phonoangiograms for these cases correlated well with predictions of greater than 5 mm or normal values for the residual lumen diameters. It is interesting to note, however, that in two of the above bruit-producing arteries of 'normal' diameter, the radiograms revealed an unusually tortuous or kinked carotid flow route (fig. 5).

The recordings of seven arteries in six individuals yielded no data compatible with turbulence but radiograms for these cases demonstrated extracranial stenosis. Four of these seven arteries were completely occluded, two had plaques associated with narrowed lumen diameters of approximately 4 mm each and the seventh case had excessive background noise on the recording. Phonoangiography is not applicable to complete stenosis where lack of flow prevents the production of bruits, or to incomplete stenoses if low flow significantly reduces bruit formation.

One hundred thirty-three arteries in 111 individuals showed negative extracranial findings on angiography and did not have any carotid bruits in their sound recordings. Transmitted heart sounds, however, could be identified in nearly all of these cases by inching the microphone from the base of the neck to the angle of the mandible and noting the decrease of signal amplitude.

Phonoangiography has been proposed as a means for following atherosclerotic lesions to determine natural history or therapy effects. We had the opportunity to record bruits during angiography of a patient with subclavian stenosis which changed in severity according to arm position (fig. 6). Bruit quality changed in parallel fashion and was correlated. Radiographic measurement of stenosis with arm extended above the head was 3.1 mm versus a phonoangiographic diameter prediction of 4.0 mm. With the arm at the side, radiographic diameter was 8.0 mm and phonoangiographic diameter 9.0 mm. In addition, two abdominal aortic bruits were recorded and compared with angiographic findings. Phonoangiography and angiographic diameters were 5.0 mm and 6.0 mm in the first case. In the second patient, they were 9.2 mm and 10.0 mm. Subclavian and aortic diameter stenosis estimations by phonoangiography employed the constant flow velocity, \( U = 50 \) cm/sec.

**Discussion**

Our results support previous publications by Lees and co-workers regarding the theory of phonoangiography and largely confirm the clinical applications they propose. First,

<table>
<thead>
<tr>
<th>Arteries</th>
<th>Patients</th>
<th>Radiographic findings</th>
<th>Bruit</th>
<th>Acoustic analysis results</th>
<th>Clinical utility of phonoangiography</th>
</tr>
</thead>
<tbody>
<tr>
<td>18</td>
<td>14</td>
<td>Stenosis present</td>
<td>present</td>
<td>Stenosis predicted</td>
<td>Phonoangiography</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
<td>Stenosis absent</td>
<td>present</td>
<td>No stenosis predicted</td>
<td>Phonoangiography of value</td>
</tr>
<tr>
<td>7</td>
<td>6</td>
<td>Stenosis present</td>
<td>absent</td>
<td>No stenosis predicted</td>
<td>Phonoangiography of potential value</td>
</tr>
<tr>
<td>133</td>
<td>111</td>
<td>Stenosis absent</td>
<td>absent</td>
<td>No stenosis predicted</td>
<td>Phonoangiography not applicable</td>
</tr>
<tr>
<td>Total</td>
<td>162</td>
<td>135</td>
<td></td>
<td></td>
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</tbody>
</table>

**TABLE 1. Clinical Results of Carotid Phonoangiography**
in regard to theory, the pseudo-sound model for isolated arterial constrictions developed by Fredberg7 should be equally applicable to sound analysis utilizing the power spectrum or to autocorrelation. Results presented in figure 3 demonstrate equivalency of the two analyses on recorded bruits.

Pseudo-sound is assumed to be a time-stationary turbulent pressure field at the artery wall.7 The flow is assumed quasi-steady at systole because the turbulent time-scale is much less than the systolic time-scale. Fluid motion in the artery is not significantly affected by arterial wall motion even though the turbulent flow field does excite the surrounding tissue. Fredberg7 assumes that the artery may be effectively replaced by a line-source of resultant forces located at a distance H below the skin surface. If L is considered to be the characteristic decay length of turbulence, then as H/L approaches infinity the artery appears as a point source with respect to the microphone. When H/L = 1, the microphone is located above the point of maximum sound intensity.

The spectrum corresponding to a bruit measured at the microphone becomes attenuated at higher frequencies of turbulence because random signals add in the mean square. Therefore, N eddies on a straight line, each of strength 1/N,
trauma indicates that records can be made in restless, tachypneic, or uncooperative patients, but only with considerable difficulty. We believe additional improvement in microphone attachment procedures and noise filtering techniques are required plus an immediate bedside presentation of results before phonoangiography will be of significant clinical value in acutely ill patients. Since cervical bruits can be rapidly identified under most conditions, an immediate rapid noninvasive estimate of underlying stenosis might lead to improved emergency medical care and further development of the method toward this goal seems indicated.

Second, we agree that phonoangiography may reduce the need for angiography in patients with identified cervical bruits. It is important to emphasize that the test is not applicable in the absence of bruits and that patients without bruits may have either no stenotic lesion or very severe stenosis. This peculiarly circular relationship between very severe stenosis and no stenosis, plus the relatively low prevalence of cervical bruits, severely limits the scope of phonoangiography as an alternative for radiographic angiography.

Last, the use of phonoangiography to follow atherosclerosis during therapy would be facilitated by increased sensitivity to small lesion change. Currently, changes in atherosclerosis during therapy are inferred by enumerating clinical episodes of ischemia. This practice allows estimates of the number of patients whose blood vessels have grown worse during treatment, but does not distinguish those whose vessels have not changed from those whose lesions may have improved. More direct vessel assessment is desirable, and to be satisfactory should be as capable of detecting lesion improvement as deterioration. We have studied an acutely changing lesion by phonoangiography and have also compared phonoangiography with radiography in static lesions. The sensitivity we find approximates that reported by Lees and co-workers. Phonoangiography
appears capable of detecting the major changes in lesions which have been observed by us and others when atherosclerosis progresses.\textsuperscript{11-18} The relatively smaller improvements in lesions observed after a therapy interval approximating one year by Barndt and co-workers\textsuperscript{19} would not seem detectable by phonoangiography unless means can be found for increased sensitivity.

Appendix

An actual autocorrelation of a carotid bruit has some minimal but nevertheless undesirable contributions due to transmitted heart sounds and background noise. The method of data processing by autocorrelation is reviewed, with a primary emphasis on the technique used to distinguish turbulence from other portions of the acoustic signal. Previous investigators have selected, on a CRT display, a discrete portion of their carotid signal for digitization and analysis.\textsuperscript{1} In this way, they exclude a part of the systolic signal where the transmitted pulse wave contribution is expected to be large. The present authors believe that the improvement in this kind of pulse wave removal can be misleading because this wave and the turbulent bruit are both often large near the leading edge of the signal. The removal of one element also tends to remove the other. In cases where a pulse wave or heart sound obviously dominates the rest of the signal, the data are misleading and are best discarded before processing.

First consider the result of processing a signal when there is minimal turbulent flow. The signal would be produced primarily by the propagated pulse wave. The signal would initially be filtered, in part by the parabolic microphone housing but mostly by the chosen electronic filter, to pass frequencies between 1 kHz and 3 kHz. The frequency response of the microphone and filter combination enhances turbulence and suppresses the large-amplitude low-frequency waves which do not contain any flow information. The period of the resultant filtered pulse wave form would, of course, be equal to the period of the heart beat as seen on the oscilloscope display, figure 1a.

The autocorrelation of the above waveform would have a full-width \( r_1 \) and have a value at zero time delay equal to the mean square of the signal as shown in figure 1b. This mean square may be approximated by \( A^2 r_1 / T \), where \( A \) equals the signal amplitude of the pulse wave and \( r_1 / T \) equals the fraction of time where the pulse wave is of significant magnitude. This autocorrelation function does not indicate whether the signal which produces it is random or periodic. The same is true of the corresponding spectral display.

Useful data are found only in the turbulent portion of the signal. Accordingly, a pulse wave's contribution to the autocorrelation is unwanted. We will consider now how a pulse wave can be taken into account in our analysis without arbitrary segregation of systole and diastole. In order to do this, we consider a second hypothetical case consisting of the ideal bruit with bursts of random turbulence and no pulse wave contained therein but nevertheless occurring periodically with each pulse wave. The oscilloscope signal would appear as in figure 1c. The amplitude of the bruit begins with a value \( B \) and falls off in a time \( T_2 \) after which the amplitude of turbulence is very low because of minimal diastolic flow. In some cases, the decaying turbulence may last a long time into diastole and may persist almost until the next bruit commences. The autocorrelation magnitude of this bruit is approximated by \( B^2 r_1 / T \) and would have a shape as shown in figure 1d. The combination of pulse wave and turbulent bruit produces an autocorrelation equivalent to the sum of the two above individual cases. The autocorrelation function, therefore, may take on a number of distinct shapes.

Shape \#1

If \( B \gg A \) and \( r_2 \gg r_1 \), or if \( B \ll A \) and \( r_2 \ll r_1 \), then the contribution to the autocorrelation is almost entirely from the turbulence and the function appears as in figure 1d. Approximately half the bruits analyzed in figure 4 (text) had this configuration.

Shape \#2

If \( A^2 r_1 \sim B^2 r_2 \), the two contributions are of the same order of magnitude. In this case, if \( r_1 \ll r_2 \), the autocorrelation is as in figure 2a. Here, the time scale applicable to turbulence is \( r_2 \) and not the axis-intersection \( r_1 \). In this instance, therefore, one is able to recognize the pulse wave's contribution to the autocorrelation and proceed by measuring the function's width at the point \( P \), where autocorrelation curvature is close to a maximum. Approximately one-third of the bruits analyzed in figure 4 (text) are of this configuration.
Figure 1. Signal analysis and autocorrelations for two hypothetical cases: a) oscilloscope trace of a signal with minimal turbulence predominated by a propagated pulse wave; b) autocorrelation corresponding to trace shown in (a); c) ideal bruit with bursts of random turbulence but no pulse wave contained therein; d) autocorrelation corresponding to trace shown in (c).

Figure 2. Autocorrelations of typical signals for cases where the pulse wave and turbulent contributions are of the same order of magnitude: a) $T_3 << T_1$, b) turbulence is of low frequency, i.e., $T_3 \sim T_1$; c) and d) cases where $T_3$ must be differentiated from $T_1$ so that time scales should be determined at point $P$ (shapes #4 and #5).
The Role of the QT Interval in the Sudden Infant Death Syndrome

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AND RICHARD R. LIBERTHSON, M.D.

SUMMARY To evaluate the role of QT interval prolongation in the genesis of the sudden infant death syndrome (SIDS), the postresuscitation electrocardiograms of 21 aborted SIDS infants were reviewed. The infants had been found apneic, cyanotic, limp and unresponsive during sleep and required vigorous physical stimulation and mouth-to-mouth resuscitation. Three subsequently experienced repeat similar episodes from which they could not be resuscitated.

THE SUDDEN INFANT DEATH SYNDROME (SIDS) is the leading cause of infant death between the ages of 1 and 12 months, and claims approximately 10,000 lives in the United States each year.1-3 Because these deaths are sudden, usually occur outside of the hospital, are observed only rarely by physicians, and have few associated autopsy findings,4-6 we still know very little about their etiology. Explanations for these deaths include asphyxia, laryngeal or bronchospasm, infection, occult endocrine, neurologic or renal disease, apnea, and autonomic dysfunction.4-11 In addition, QT interval prolongation with fatal ventricular arrhythmia has been proposed as a cause for SIDS.12-17 However, because SIDS victims themselves rarely have electrocardiographic evaluation before death, recent workers have examined relatives of SIDS infants because it is known that QT prolongation in some families is genetically transmitted.18-20 Based on this indirect approach, these studies suggest a causal relationship between QT prolongation and the SIDS.21,22 In this report, we examine the postresuscitation QT intervals of aborted SIDS infants, including three who subsequently died from actual SIDS.

Methods

Between 1974 and 1976, 21 survivors of the aborted SIDS were referred to the Massachusetts General Hospital

References

Phonoangiography by autocorrelation.
R M Rosen, S P Parthasarathy, A F Turner, D H Blankenhorn and E J Roschke

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