Effects of Surrounding Tissue on the Sound Spectrum of Arterial Bruits in Vivo

ARNOLD MILLER, M.D., ROBERT S. LEES, M.D.,
J. PHILIP KISTLER, M.D., AND WILLIAM M. ABBOTT, M.D.

SUMMARY Turbulent flow distal to arterial stenoses produces bruits with a characteristic sound spectrum, analysis of which has permitted accurate non-invasive assessment of the residual lumen diameter of the stenosis in the case of the human carotid artery. In contrast, investigators working with in vitro elastic models of arteries or with excised vessels have reported finding mainly resonant spectra of bruits recorded distal to stenoses. We have studied the effects of turbulent flow on the sound spectrum produced at the arterial wall and the influence of surrounding tissue on this spectrum. Aortic, carotid, and femoral stenoses were produced in dogs by external banding of the arteries with 5mm wide Teflon bands. Recordings of bruits made directly on the vessel wall had a sound spectrum made up of 2 components, one due to turbulent flow, and the second to a superimposed resonant spectrum from arterial wall vibration. This was true of 3 kinds of vessels studied. The effects of surrounding tissue on the sound spectrum of arterial bruits was shown by comparing the spectra of bruits recorded directly on the vessel wall, on the freshly closed wound and on the healed wound. The sound properties of the artery in situ are very different from those of exposed or excised vessels or elastic tubes. Although intravascular turbulence may be accurately appreciated at the skin surface, arterial wall resonance in the intact animal is extensively damped by the normal coupling of the artery to its surrounding tissue.

Methods Two experimental animal models were used. With the first model we investigated the effects of poststenotic turbulent blood flow on the sound spectrum of the bruit recorded directly on the exposed vessel in vivo. With the second model we studied the effects of the surrounding tissues on the characteristics of this spectrum.

Study 1

This animal model has also been used to quantitate the relationship of blood flow to the sound produced by a stenosis and is described in detail elsewhere. Ten adult mongrel dogs were anesthetized with sodium pentothal 20–30 mg/kg. Twelve cm of the jugular vein were carefully excised and all tributaries ligated. The femoral arteries and veins in both thighs were exposed. Using the jugular vein as a free graft, arteriovenous fistulae were created bilaterally between each femoral artery and the adjacent femoral vein by end-to-side suture. The abdomen was then entered through a midline incision and the abdominal aorta exposed. A stenosis sufficient to cause a bruit was created by the external application and suturing of a 5 mm wide Teflon band around the aorta approximately 8 cm above the bifurcation. An electromagnetic flow meter, used for relative flow and not quantitative measurement, was placed 6 cm distal to the stenosis. Screw clamps were applied to the A-V fistulae, enabling flow
through the fistulae and through the stenosis to be modified in a graduated and controllable fashion.

Bruits produced at each of 15–18 different flow rates were recorded and analyzed as described below.

Study 2

Eight adult mongrel dogs were anesthetized with sodium pentothal, 20–30 mg/kg and under sterile conditions the femoral and/or carotid arteries were exposed. By the external application of a 5 mm wide Teflon band, 20 stenoses of varying diameters sufficient to cause bruits were created. The bruits produced were recorded as described below but in this study the microphone was applied directly to the vessel wall. Recordings were made at various sites both proximally and distally.

The wound was closed in layers, taking care to mark the site of the stenosis on the skin surface with a silk suture. After wound closure, the bruit was again recorded, this time on the skin surface of the freshly closed wound.

The wound was then allowed to heal. Six to 12 weeks later, the dogs were reanesthetized, the same bruits recorded, and the results compared.

Recordings, Analysis and Interpretation

Bruits were recorded with a piezo-electric displacement transducer (Hewlett-Packard 21050 B) whose frequency response had been precalibrated, and stored on magnetic tape (Tandberg No. 3000x) as previously described. Two recordings of each bruit were made, one immediately distal to the stenosis and the second 3–4 cm further distal. Initially, the transducer was placed directly on the aorta, but because of damage to the sensitive microphone from aortic wall vibration, subsequent recordings were made over a thin Penrose drain filled with ultrasonic gel placed directly on the aorta. This filtered only the coarse low-frequency vibrations of the arterial wall. Frequencies over 50 Hz were unaffected as determined by multiple trials with and without the gel-filled drain.

The recorded bruits were played back through a preamplifier (Princeton Applied Research No. 113) into a high speed A/D converter (Analogics AN5800) and then into a minicomputer (Data General Nova 1220), for Fourier analysis of the sound spectrum of the peak systolic segment, as described previously.

The spectrum was displayed on a cathode ray oscilloscope and a permanent copy made.

Results

The characteristic sound spectrum of turbulent blood flow has been previously described and is shown in figure 1. The spectrum is relatively flat up to a discrete peak after which intensity falls off rapidly with increasing frequency.

A series of typical spectra of the bruit of abdominal aortic stenosis made at several different blood flow rates in model 1 is shown in figure 2. At lower flow rates, the intensity increases with frequency to a single discrete maximum, beyond which it falls off with a characteristic slope, as frequency increases further. The frequency at which the peak amplitude occurs is the "break" frequency, $f_0$.

![Figure 1](http://example.com/figure1.jpg)

**Figure 1.** Tracing of the sound spectrum of a typical post-stenotic arterial bruit at peak systole. The intensity of sound increases with frequency to a single discrete maximum, beyond which it falls off with a characteristic slope, as frequency increases further. The frequency at which the peak amplitude occurs is the "break" frequency, $f_0$.

![Figure 2](http://example.com/figure2.jpg)

**Figure 2.** Tracings of the sound spectra of aortic bruits produced by altering the flow through a fixed stenosis. Arrows indicate the "break" frequency, $f_0$, of turbulent flow. Note the appearance of multiple peaks, over a wide frequency range, with increasing flow rates.
velocities, where turbulence is less pronounced, the spectrum is typical for turbulent flow, with a smooth pattern and a clear cut break frequency. As flow velocity increases, multiple peaks appear over a wide frequency range between 100-1500 Hz. The frequency of these peaks, once they are apparent, does not appear to change with flow velocity. Change with increasing flow velocity is reflected mainly in their increased intensity. The peak peculiar to the turbulent spectrum increases in frequency, as expected, with the progressive increase in flow velocity. However, in the recording from the exposed aorta, this peak becomes increasingly difficult to distinguish from the other peaks.

Figure 3 shows that once a certain degree of turbulent flow is reached, as in this animal with a very tight abdominal aortic stenosis, to increase the flow further has little influence on the number, shape and intensity of the peaks in the sound spectrum.

With experimental model 2, the relationships among microphone locations with respect to a stenosis of the carotid or femoral artery, the presence of overlying tissue and the characteristics of the bruit spectrum were examined. Figure 4 is a typical example of the spectra obtained at various sites in relation to a carotid stenosis. Within 3 cm proximal and 4 cm distal to the stenosis, the frequency and configuration of the various peaks did not vary appreciably. The intensity did change, however, and was maximal within 1.5-4 cm downstream from the stenosis.

The effects of the surrounding tissues on the spectrum of carotid and femoral bruits are exemplified in figure 5. Multiple peaks are readily seen on the "open" spectrum, where the bruit has been recorded with the microphone directly applied to the dog's carotid artery. Many of these peaks disappear when the bruit is recorded on the skin surface immediately after the incision is sutured, but the tracing is still multi-peaked and irregular. Twelve weeks later, with the wound healed and the artery firmly tethered by sur-

Figure 3. Tracings of the sound spectra of a tight abdominal aortic stenosis in a dog, with increasing flow rates. Once a certain degree of turbulent flow is reached, any further increase in flow has little influence on the number, shape and intensity of peaks in the sound spectrum.

Figure 4. Relationship between microphone location on the exposed carotid artery and the spectrum of sound produced by a stenosis in this vessel. Note that maximal intensity occurs either immediately distal or within 1.5-4.0 cm downstream from the stenosis.
SOUND SPECTRUM OF ARTERIAL BRUITS/Miller et al.

The specific location of these resonant peaks, in particular the highest frequency peak, suggests that they are dependent on the properties of the particular vessel, including its location in the body and its thickness. In the dog aorta the highest peak had a frequency of between 1000–1500 Hz as compared to the carotid and femoral arteries, where the peak appeared at 800–1000 Hz with some overlap between the 2 groups. This is similar to the in vitro findings of Kirkeeide et al. who showed the specific location of the highest frequency peak to be solely dependent on latex tube conditions, wall thickness and applied axial tension, and is also in keeping with their suggestion that the location of these peaks is the result of resonant vibrations of the tube or, in our case, the artery itself. Determinations of the resonant spectrum of an artery or arterial prosthesis may be useful in defining its mechanical and structural properties, allowing comparison among the various vessels, normal and diseased arteries, and arterial substitutes.

Perhaps the most significant findings from our animal experiments are the effects of surrounding tissue on the sound spectra of bruits recorded at the skin surface. On the exposed vessel the 2 spectra described above are seen, with the resonant spectrum often obscuring the turbulent spectrum. Wound closure results in partial damping out of the resonant spectrum and healing leaves a clear, smooth, characteristic turbulent spectrum which is easy to interpret. We conclude that a normally tethered artery does not usually resonate and spectral analysis of a stenotic bruit recorded at the skin surface shows ordinarily only the typical turbulent spectrum. Incomplete tethering and damping out of the resonant spectrum would account for distortions and artifacts occasionally seen, which sometimes make difficult interpretation of bruit spectra in patients with carotid artery stenosis.

In our studies, the greatest intensity of these resonant peaks, when recorded on the exposed artery, occurred within 1–4 cm distal to the stenosis. This is where the turbulent jet from the stenosis may be expected to impinge on the arterial wall and is the...
region where post-stenotic dilatation occurs. Roach and colleagues\textsuperscript{6,13} have shown that isolated segments of arteries subjected to various external frequency vibrations become more distensible. They have postulated that post-stenotic dilatation may be due to a type of structural fatigue caused by vibration of the wall secondary to turbulence in the fluid distal to the stenosis. However, they were unable to record these vibrations \textit{in vivo}. Our studies confirm their hypothesis and have shown that apparently resonant vibration of the arterial wall due to post-stenotic turbulent flow does occur, at least in untethered vessels. Post-stenotic dilatation may be most prominent, for this reason, in vessels which are normally incompletely tethered, for instance, the main pulmonary artery and ascending aorta distal to the pulmonic and aortic valves, and the epicardial portion of the coronary arteries.

References

8. Fredberg JJ, Lees RS, Dewey CF Jr: How to listen to your arteries (or what your doctor would hear if he were a fluid dynamicist), paper no. 70–144. Proceedings of AIAA Aerospace Sciences Meeting, New York, 1970
Effects of surrounding tissue on the sound spectrum of arterial bruits in vivo.
A Miller, R S Lees, J P Kistler and W M Abbott

Stroke. 1980;11:394-398
doi: 10.1161/01.STR.11.4.394

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1980 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/11/4/394

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/