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.

ALTHOUGH AUSCULTATION of arterial bruits has been taught since the time of Laennec, only recently has quantitative information been derived concerning the extent of arterial disease. Lees and Dewey1 in 1970 showed that flow through a significant arterial stenosis resulted in a spectrum of pressure fluctuations at the surface of the skin remarkably similar to wall pressure fluctuations in fully turbulent pipe flow at a high Reynolds number, and that quantitative information about the size of the residual lumen at the stenosis could be obtained by spectral analysis of the bruit recorded at the skin surface overlying the artery. In later studies, Lees and collaborators showed that accurate clinical diagnosis of the extent of carotid stenosis could be made non-invasively by quantitative analysis of bruit spectra.2 4 The analytical method depends upon the recognition of a single frequency peak beyond which sound intensity drops sharply with increasing frequency.

By contrast, Boughner and Roach showed that isolated arteries studied ex vivo resonate when excited by a sound stimulus at certain characteristic frequencies.3 Kim and Corcoran6 showed that turbulent flow in latex tubes produced 2 spectra superimposed on one another — a turbulent spectrum caused by disturbed fluid flow and a resonant spectrum resulting from the natural frequency of the tube itself, which was set in vibratory motion by the turbulent flow.

The present study was designed to investigate whether turbulent blood flow in arteries in situ produces arterial wall resonance in vivo and to study the effects of the surrounding tissues on the characteristic sound spectra produced.

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.7 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
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.

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 5. Tracings of the sound spectra of bruits recorded on the exposed carotid artery (open) and on the skin surface immediately after wound closure (closed), and 12 weeks later (healed). The turbulent peak at 325 Hz is followed by a characteristic falloff which is completely obscured by the resonant spectrum of the exposed vessel. Most of the resonance is damped when the wound is closed and almost all of it when the wound is healed.

rounding tissue, the sound spectrum recorded at the identical location is smooth, with a peak and characteristic falloff which is typical for turbulent flow associated with a bruit. The break frequency is recognized without any difficulty. This sequence of events occurred with each of the 20 vessels so studied.

Discussion

Our results show that the spectra of bruits produced by arterial stenosis, when recorded in vivo on the exposed artery, are different from the spectra produced by the same stenosis when recorded at the skin surface over the healed incision. The spectrum recorded over the exposed vessel is composed of the sum of 2 components. One is the spectrum related to turbulent flow distal to the stenosis and the other the resonant spectrum of the arterial wall which is set in motion by the turbulent blood flow within it. When the incision over the stenotic vessel is allowed to heal, the tethering produced by the surrounding tissue prevents or strongly damps arterial wall resonance and the relatively intact turbulent spectrum can be appreciated at the skin surface.

The turbulent spectrum is characterized by a gradual increase in intensity with increasing frequency to a discrete frequency-intensity peak followed by a rapid falloff in intensity. Analysis of such spectra is the basis of a quantitative non-invasive method for determining the residual lumen diameter of an arterial stenosis when a bruit is present.

The resonant spectrum, by contrast, is characterized by the presence of multiple intensity peaks over a wide frequency range. This spectrum derives from the vibration of the arterial wall itself, which was similar in our in vivo studies and in the ex vivo studies of Boughner and Roach. These findings have been confirmed by others in experimental models both in vitro and in vivo.

In our studies, increasing turbulent flow was associated with an increase in the amplitude of the resonant peaks to a maximum while the frequency of the peaks did not change.

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. 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.
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 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.

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