Arterial Wall Vibration Distal to Stenoses in Isolated Arteries of Dog and Man

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ABSTRACT
It has been suggested that poststenotic weakening of arterial walls is caused by arterial wall vibration and structural fatigue initiated by pressure disturbances in turbulent blood flow. We examined the frequency spectra of wall vibration downstream from experimental arterial stenoses and compared them with the natural resonant vibration characteristics of the artery walls. Spectra of arterial wall displacement of isolated perfused dog and human arteries were measured with a noncontacting capacitance transducer during conditions of fluid flow through the artery; resonant vibration characteristics were determined during pure sine and random sound stimulation of the artery walls in the absence of flow.

It is concluded that fluid flow through isolated arteries with an experimental stenosis excites the artery wall to vibrate over a wide range of frequencies within which are discrete frequencies that coincide with the resonant frequencies of the artery wall.

ADDITIONAL KEY WORDS poststenotic dilatation resonance vibration transducer structural fatigue frequency analysis noise stimulus

In man, arterial dilatation downstream from a stenosis occurs in many arteries and with stenoses of both intrinsic and extrinsic origin, e.g., with aortic and pulmonary valvular stenosis, coarctation of aorta and pulmonary arteries, distal to atheromatous plaques in the carotids, femorals, renals and other medium-sized arteries (1). The exact cause of the dilatation has been puzzling; De Vries and Van den Berg (2) showed that the fluid static pressure is always normal or lower than normal distal to a stenosis. Roach (1, 3), in a study of experimentally produced poststenotic dilatations, both in vivo and in vitro, found an increased distensibility of the artery wall distal to the stenosis, provided that the stenosis had produced "distal turbulence" as defined by the presence of a thrill and bruit. She concluded that the increase in distensibility was due to a change in elastin fibers and that there was some correlation between the amount of increase in distensibility and the intensity of the murmur caused by the stenosis. This implies that the stenosis produces turbulent flow and associated pressure fluctuations which in turn cause the artery wall to vibrate, thus leading to a weakening of the elastin fibers in the artery wall. This process would appear to be akin to the phenomenon of structural fatigue of materials under dynamic loads. This comparison with structural fatigue was first made by Holman (4), who, in addition to studying poststenotic dilatation in humans, produced a similar phenomenon in thin-walled rubber tubes. Bruns et al. (5) further showed that a dilatation could be induced in thin-walled rubber tubes distended with water that was artificially vibrated (i.e., pressure fluctuated) in the absence of flow.
In the present experiments, an attempt was made to analyze arterial wall vibration downstream from experimental stenoses of isolated perfused arteries. The vibration so produced was also compared with the natural resonance characteristics of the artery as determined by external stimulation of the artery with sound energy.

Methods

Perfusion of Isolated Arteries

Human external iliac and dog external iliac and femoral arteries 3.5 to 6.5 cm long were perfused at room temperature with 0.9% saline solution containing merthiolate 1:10,000, a solution which preserves elastin and collagen tissue (6). A diagrammatic sketch of the perfusion apparatus and flow system is shown in Figure 1.

The artery was perfused in the acrylic chamber by recirculation through the system, in which the lower reservoir was maintained at a constant height above the artery (130 cm). The net perfusion pressure, and therefore flow rate, was controlled by varying the height of the upper reservoir. Flow rate was constant during each experiment and was measured by timing a given quantity of fluid in a graduated glass beaker; flow rates chosen were comparable to those at peak of systole in vivo.

The artery was mounted in the acrylic chamber between two thin-walled stainless steel tubes which were sealed through the wall of the acrylic chamber and connected to the perfusion system. An external stenosis was imposed on the artery by an acrylic ring which sealed into a hole in the center wall of the chamber by an O-ring. Acrylic rings with various internal diameters were made and fitted over each test artery. The sizes of stainless steel tubes and corresponding fittings were chosen so that in each experiment the internal diameter of the tube was as close to the lumen of the artery as possible. Once the perfusion was started and all air bubbles removed from the system, the artery was stretched to its in vivo length in dog arteries and until there was slight longitudinal tension in human arteries. The acrylic chamber was then filled with saline-merthiolate solution, and the level of this fluid was then reduced until two-thirds of the artery was exposed above the fluid during wall displacement measurements. The internal geometry of each artery was measured in each experiment by making a cast of the artery lumen with Silastic (Dow Corning Elastomer no. 385 diluted with Medical Fluid no. 360). The percent stenosis (percent ratio of the internal diameter at the stenosis to the internal upstream artery diameter) varied from 37 to 63. The Reynolds numbers upstream from and at each stenosis were calculated; these ranged from 777 to 2650 and from 1320 to 6180, respectively.

Measurement of Wall Displacement During Flow

The displacement transducer was a noncontacting capacitive type (Bruel & Kjaer, type MM0004) with a flat frequency response to 200,000 Hz. The air gap between the transducer and the test object (which was grounded) was charged by a polarization voltage (300 v) from a cathode follower (B & K type 2615) to which the transducer was connected. The loading of the artery wall by this transducer (static attraction) for the gap distances employed (1.5 to 3.0 mm) was much less than 6 mg. The complete test

Diagram of perfusion of isolated arteries and measurement of wall displacement.
ARTERIAL WALL VIBRATION

For Cable

**FIGURE 1**
Diagram of constant amplitude sound stimulation and wall displacement measurement of artery.

The system is shown diagrammatically in Figure 1. The output of the transducer, via the cathode follower, was fed to a constant percent narrow band frequency analyzer (B & K type 2107), from which the output was fed to a level recorder (B & K type 2305). This level recorder acted effectively as a recording voltmeter. The motor of this recorder also drove the sweep of the frequency analyzer, which could be synchronized with the speed of the frequency-calibrated paper. In this way, an amplitude-frequency recording was obtained.

A static calibration of the transducer was not possible; the relative amplitude of the vibration has therefore been expressed in terms of the voltage level of the vibration signal at the input of the frequency analyzer.

In all recordings of this and subsequent tests, the experimental test section was mounted on a massive concrete slab isolated by air springs from any pumping and extraneous building vibration effects.

**CONSTANT AMPLITUDE SOUND STIMULATION OF THE ARTERY WALL IN THE ABSENCE OF FLOW**

Sound was produced by driving an inexpensive loudspeaker with the output of a sine-random generator (B & K type 1492), as shown in Figure 2. The sound output of the loudspeaker was directed at the artery which was located and supported in the same manner as during wall vibration measurements described above by sealing a plastic funnel over the cone of the loudspeaker. A plastic Y-tube was connected to the narrow opening of the funnel, and sound pressure at both openings of the Y-tube was similar when tested with two identical microphones. One arm of the Y-tube was directed at the artery wall adjacent to the capacitive transducer. The other arm was used to control the output of the signal generator so that a constant sound pressure was produced at the openings of the Y-tube as the frequency range was varied. To do this, a microphone (B & K type 4131) was placed in close apposition to the opening of the second arm of the Y-tube. The output of the microphone was fed via an amplifier (B & K type 2603) to the "compressor" circuit of the sine-random generator. This circuit acted to monitor and control the sound pressure at different frequencies at the two branches of the plastic Y-tube, attenuating the output of the signal generator as the loudspeaker (and system) passed through frequencies at which resonance occurred.

With this apparatus, the arteries were stimulated by pure (single frequency) sine-wave sound swept through frequencies from 20 to 1000 Hz while the displacement of the artery wall was recorded by the capacitance transducer via the 2603 amplifier without any filtering. Three arteries, in a separate test, were exposed to a random noise stimulus with a bandwidth of 300 Hz and a center frequency of 150 Hz. This stimulus was maintained constant while a frequency analysis of the arterial wall displacement was performed.
was carried out with the 2107 frequency analyzer. During both these tests there was no flow through the artery; it was, however, internally pressurized by connection to the upper saline reservoir.

**Results**

Arterial wall displacement downstream from a stenosis was analyzed with the capacitance displacement transducer (Fig. 1) during constant flow through isolated arteries (two femoral and two common iliac arteries from dogs and seven human external iliac arteries). Figure 3 shows a relative amplitude-frequency recording from an experiment on a human external iliac artery. The stenosis reduced the internal diameter of the artery to 45% of the upstream diameter. The flow rate of saline through the artery was 1000 ml/min, the Reynolds number upstream of the stenosis was 2650 and at the stenosis was 5940. The electrical noise level of the instrumentation is not shown in this figure but was recorded in every experiment. In this experiment there was some 120 Hz of noise; however, it was not synchronous with the 130-Hz peak and its level was 30 db (i.e., 32 times) lower than that of the test level at 120 Hz. It can be seen that there was little variation in the amplitude of vibration at the frequencies between 20 and 90 Hz. The amplitude then rose to a peak at 130 Hz, after which it decreased rapidly. There were two further but smaller peaks at 310 and 550 Hz. This record is typical of that obtained in the other experiments except for a slight variation in the number and position of the peaks from artery to artery. In the setting up of individual experiments at this point, it was noted that the amplitude of vibration could be increased by tightening the ring (increasing the stenosis, provided flow rate was little affected) or by increasing the flow rate for a given stenosis.

In 9 of the 11 experiments the distal segment of the artery was stimulated with pure (single frequency) sine sound, swept through the frequency range of 20 to 1000 Hz (as shown in Figure 2). During this stimulation there was no flow of fluid through the artery, but the pressure of the fluid in the artery was equal to the upstream pressure used during flow. The result of stimulation by pure sine sound is shown in Figure 4; the artery used in this part of the experiment was the same as that used in the constant-flow test.
Comparison of peak amplitude frequencies during sound stimulation (ordinate) and during flow (abscissa).

FIGURE 6
Resonant vibration of wall of same artery as Figure 3 (45% stenosis, no flow) during sound stimulation with random noise of 300 Hz bandwidth and center frequency of 15 Hz. Units on ordinate are voltage level of signal at input to frequency analyzer.

of Figure 3. A number of resonance peaks are apparent, three of which are at frequencies similar to the frequencies of the peaks in Figure 3, namely, the two large peaks at 130 and 290 Hz and the small peak at 550 Hz.

Thus amplitude peaks with frequencies similar to those occurring during flow through a stenosis occurred under these conditions. Because the amplitude of the sound stimulation was maintained at a constant level at all frequencies used, amplitude peaks under these conditions must be due to resonant vibration of the artery wall. The frequencies of these amplitude peaks are therefore resonant frequencies of the artery wall. The frequencies of the amplitude peaks during flow through stenoses (produced in the nine arteries stimulated by single-frequency sine sound) are compared with the respective resonant frequencies in Figure 5. In this figure the frequency of each amplitude peak of wall vibration during flow is plotted against the frequency of the closest amplitude peak during sound stimulation, or, in other words, the closest resonant frequency for that artery. It can be seen that there is good correlation between the frequency of the amplitude peaks and the resonant frequencies over the lower
frequency range examined, but at the higher frequencies the correlation is less. The correlation coefficients for the first three groups were significant: \( r = 0.93, P < 0.001; r = 0.98, P < 0.001; r = 0.89, P < 0.05 \), respectively. These results suggest that, during flow simulating that at systole, amplitude peaks in the vibration of the arterial wall distal to stenoses are due to resonant vibration of the artery wall rather than to stimulation at one particular frequency more than another.

A further comparison of artery wall vibration downstream from a stenosis (with flow) with artery response resulting from a different form of sound stimulus (with no flow) was made in three arteries. Although there was no flow through the artery during this stimulation, fluid pressure was again kept the same as the upstream pressure used during flow. The sound stimulation was random noise, with a center frequency of 150 Hz and a bandwidth of 300 Hz. This stimulus was held constant, and a frequency analysis of wall vibration was carried out as shown in Figure 2. The result of one of the experiments, again using the same artery as in the test of Figure 3, is shown in Figure 6. Although the amplitude is not constant between 20 and 90 Hz and there is a peak between 50 and 60 Hz, the peaks at 130 and 280 Hz correspond closely with similar peaks in the frequency analysis during flow of Figure 3. There is no peak at 550 Hz, but this is not surprising when the upper frequency limit of the stimulus was 300 Hz. In the other two experiments of this type, there was also good correlation between the peaks during sound stimulation and those during flow \( r = 0.99, P < 0.001 \). This suggests that the wall vibration distal to a stenosis during flow comparable to that at systole is the result of stimulation of the artery wall by wide-band random energy, as would be expected from fluid flow that is turbulent.

The Reynolds numbers based on artery lumen upstream from the stenosis ranged from 777 to 3350 for the arteries tested. The critical Reynolds number for transition from laminar to turbulent flow in tubes is often quoted as 2000, although this depends on various factors such as upstream approach conditions, tube roughness, etc. In any event, the validity of defining a critical Reynolds number for the circulatory system, in which flow is pulsatile, is doubtful—especially for pulsatile flow through channels of nonuniform caliber like the blood vessels. Misner and Rushmer (7) have shown that turbulence occurs downstream from a constriction at Reynolds numbers as low as 800; Roush (8) has detected turbulence distal to a stenosis at a Reynolds number of 90, using dye streaks as a tracer.

Production of dilatation of the artery wall distal to stenoses was examined in six of the previous experiments, using the method as outlined by Roush (3, 9); tension-radius curves were plotted both before and after exposure to turbulent flow for 12 to 48 hours. There was no significant displacement of the curves and therefore no suggestion of an increase in distensibility of the artery wall. In a further 15 experiments in which wall vibrations were not analyzed, no evidence of artery dilatation, as determined by diameter measurements using a 30X traveling microscope, was found after the sections distal to stenoses had been exposed to up to 72 hours of vibrations due to turbulent flow.

Discussion

These experiments show that when saline is perfused through isolated arteries with an experimental stenosis at rates comparable to the peak systolic flow through these arteries (10) vibrations of the artery wall are excited immediately downstream from the stenosis. The frequency spectrum of these vibrations showed amplitude peaks, whose number and frequency position varied from artery to artery, superimposed on a wide band of vibration extending from 20 to 1000 Hz. The frequency of these amplitude peaks corresponded closely to resonant frequencies when the artery was externally stimulated by wide-band sound energy in the absence of flow. These findings suggest that flow through isolated arteries with an experimental stenosis excites the artery wall to vibrate over a wide band of frequencies, which include resonant frequencies of the artery wall.
The vibrations are transverse displacements of the artery wall; no evidence as to the mode of vibration has been obtained. Thus it is not possible to decide whether these vibrations are purely extensional (a radial mode) or bending in nature. The absolute size of the vibrations can only be estimated because an in-situ calibration of the transducer was not possible. Calculations based on the specifications of the transducer suggest that the vibrations are of the order of 0.01 mm peak to peak.

On the basis of experiments (11) in which the fluid particle movement downstream from stenoses in acrylic tubes has been correlated with fluid pressure fluctuations at the wall, it has been suggested that fluid pressure fluctuations resulting from shear action in turbulent flow stimulate arterial wall vibration. In the present experiments no attempt was made to measure such pressure fluctuations at the artery wall. However, we believe that the results give indirect evidence on the nature of the stimulus. For example, it was possible to produce a vibration pattern of the artery wall which had a frequency spectrum similar to that occurring during flow through the experimental stenosis by stimulating the artery with a relatively wide-band random noise energy (Figs. 3 and 6). This suggests that the arteries were being stimulated by a relatively wide-band random pressure during flow (as is characteristic of turbulent flow). This indirect evidence of the nature of the flow and the stimulus to the artery wall downstream from a stenosis is supported by pressure measurements in model experiments using acrylic tubes (12, 13) and in in-vivo experiments on femoral arteries of anesthetized dogs (12). The frequency spectra of the pressure fluctuations at the wall downstream from stenoses were examined in both these situations. In both the spectra were relatively wide-band and without discrete amplitude peaks; there was no evidence that the artery was being stimulated at discrete frequencies such as would occur in vortex shedding (5).

Vibration at resonance is a situation in which relatively high strains can be produced by relatively low forces. It is thus a situation in which structural damage is likely to occur. Roach and Harvey (9) reported evidence that a dilatation could be produced downstream from a stenosis in isolated dog and human arteries by perfusion of saline through them for periods of up to 72 hours. In the present experiments we could not detect significant changes in the mechanical properties of the artery wall during such a time course. We have no direct explanation for this lack of agreement, although it may have been due to a difference in the technique of measurement or imposed conditions; however, the fact that vibrations occur at resonant frequencies of the arterial wall increases the possibility that wall damage could occur as a result of structural fatigue.

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