Effect of Low Frequency Vibration on the Arterial Wall

By Derek R. Boughner and Margot R. Roach

ABSTRACT

Poststenotic dilation is accompanied by alteration of the elastic properties of the arterial wall in the region where a murmur occurs. The low frequency vibrations contained in a murmur may cause the changes observed. Isolated human external iliac arteries were distended with 100 mm Hg pressure and vibrated with specific frequencies from 30 to 400 Hz. When dilation occurred, the radius increased at a rate of 7.1 ± 3.4 SD % per day. However, for each artery, dilation tended to occur with one frequency in preference to others. The amplitude, as long as sufficient to produce vibration of the wall, was not important in determining this tendency. The older the artery the higher the frequency necessary to produce dilation. Young vessels, under 45 years, responded best to frequencies <100 Hz, and vessels 45–60 years old responded to frequencies 100–200 Hz and older vessels, >60 years, dilated with frequencies >200 Hz. The elastic diagram of the vessel after dilation showed the major change to be in the elastin component of the arterial wall rather than the collagen. These findings correspond to those seen in a vessel displaying poststenotic dilation.

KEY WORDS poststenotic dilation elastic properties elastin arterial age changes murmurs

■ Poststenotic dilation was first described over 100 years ago (1), yet the mechanism of its formation has never been completely explained. Basically, two factors determine the diameter of a given vessel: the distending pressure acting perpendicularly to the wall in all directions and the elastic properties of the wall acting circumferentially to resist this distending force. DeVries and van den Berg (2) demonstrated that the intraluminal pressure beyond a stenosis is always normal or lower than normal. Roach (3), however, showed that the elastic diagram for the segment of artery distal to a stenosis was altered when compared to the proximal segment and that this alteration allows the vessel to assume a greater diameter for a normal intraluminal pressure.

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Histological examination of the dilated areas of arteries distal to a stenosis has resulted in conflicting opinions on the presence or absence of changes (3, 4). The variety of interpretations may in part be due to differences in vessel age but, more importantly, no vessels were examined when distended and fixed at physiological pressures. Consistent abnormalities of structure and function would more likely be visible with this method. However, it is also possible that the critical changes are not gross enough to be visualized by light microscopy.

Intravascular turbulence beyond a stenosis produces the murmur and palpable thrill which invariably accompany poststenotic dilation (5). The random motion of the intravascular fluid produces pressure fluctuations which vibrate the vessel wall (6, 7), and this energy is transmitted in part along the arterial wall as transverse vibrations (8). The alteration of the vessel wall elastic properties found distal to a stenosis might be caused by these vibrations.

The suggestion that vibration can alter the

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physical characteristics of human and animal tissue is not entirely new. For example, Gersten (9) demonstrated that ultrasound, with a frequency of 1 mHz, decreased the elastic modulus of collagen in a strip of tendon. Arterial segments have been subjected to random low frequency vibrations with the production of changes in the elastic diagram similar to those found in a dilated artery (3). We therefore set out to determine whether specific low frequency vibrations, such as those found in a murmur, could be responsible for the altered arterial elastic properties distal to a stenosis. Since previous work had been done on in vitro preparations of human external iliac arteries (10), these vessels were chosen for the study.

Methods

In artificially stenosed, in vitro preparations of human external iliac arteries, Roach and Harvey (10) produced poststenotic dilation and showed its association with turbulence and a murmur. Foreman and Hutchinson (11) could not reproduce this, but because of asymmetry and parallax, their use of a traveling microscope could have missed the small changes in radius produced. To determine whether specific vibrations could create these alterations, human external iliac arteries were obtained at autopsies and immediately immersed in a solution of 1:10,000 aqueous merthiolate in normal saline which had an osmolarity of approximately 250 milliosmol/liter. These were stored in a refrigerator at 4°C until use. This solution has been previously shown to

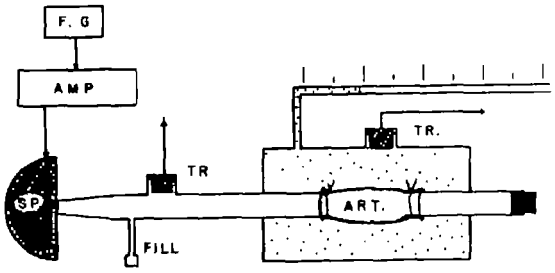

FIGURE 1

Apparatus used for vibrating arteries. Arteries were mounted on solid plastic tubing, enclosed in a plethysmograph and filled from a variable pressure reservoir. Vibrations were produced within the vessels by a loudspeaker (SP.) *driven by an amplifier* (AMP.), *which received a signal from a frequency generator* (F.C.). *Two transducers* (TR.) *monitored the frequencies inside and outside the arteries*

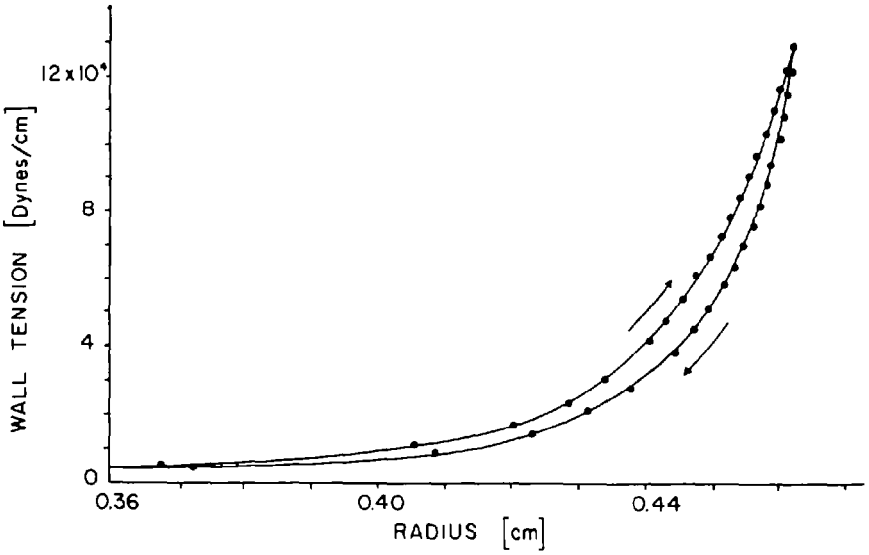

FIGURE 2

Tension-radius diagram for an external iliac artery (age 52) as calculated using the plethysmograph and the law of Laplace. Note the narrow hysteresis loop indicating viscous properties of the wall. Arrows indicate raising and lowering of pressure, total time elapsed approximately 2 minutes.

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preserve the static elastic properties of an artery for several days (12), and in our experience a normal tension-length curve could still be obtained on the tenth day after removal.

MEASUREMENT OF DILATION

Most arteries were used within a few hours of autopsy and were mounted in a closed chamber (plethysmograph) containing the same merthiolate solution. The chamber was open to atmospheric pressure along a calibrated tube (Fig. 1).

The artery was filled from a variable pressure reservoir and the volume of the artery could be measured for any given pressure by fluid displacement along the calibrated tube. Using the law of Laplace as modified for arteries (13) ($T = P \times R$, where T = wall tension, P = intraluminal pressure and R = vessel radius). An elastic diagram (Fig. 2) was constructed for the arterial wall before and after vibration. The artery was distended from 0 to 300 mm Hg pressure several times before commencing measurements. This provided a stable and reproducible curve (14). The graph plotted was a tension-radius curve not a stress-strain diagram. Since the vessel was enclosed in the plastic, fluid-filled chamber, the opening pressure for each artery could not be assessed with sufficient accuracy to give a value of the radius at zero wall tension. Thus the exact radius of the vessel at each pressure was used rather than strain. In addition, wall thickness at the various pressures could not be evaluated, so wall tension (dyne/cm) was used rather than wall stress (dyne/cm^2).

GENERATION OF VIBRATION

One end of the artery was mounted on a rigid plastic tube connected directly to a loudspeaker (Fanon HOA-5A-8) with a plastic-coated diaphragm. The other end was attached to a rigid tube closed distally by a soft rubber plug. The tubing on which the vessel was mounted had terminal "lips" over which the vessel was slipped and behind which it was tied. This lip assured that the vessel when distended maintained a shape as close to cylindrical as possible so that end-effects which might alter the application of Laplace's law were avoided. Measurements of vessel length were made between these easily visible bulges. The mounted vessel length was made to approximate the in vivo length as closely as possible. Excision of the external iliac artery produced little measurable change in the vessel length (about 1%–2%). The artery was therefore measured with a ruler before mounting and, when mounted under a slight degree of longitudinal tension, it closely approximated the in vivo length.

The loudspeaker used to vibrate the artery was driven by a Pako amplifier which received a signal from a frequency generator (Heathkit Model IGI8). The magnet and coil were immersed in fluid under identical pressures to allow the diaphragm to move when exposed to the high pressures within the artery. This fluid was circulated and therefore also acted as a coolant for continuous operation of the speaker.

The frequency response curve (Fig. 3) shows the calculated amplitude output of the loudspeaker for each frequency setting. This graph was constructed by setting a specific amplitude input for the speaker at 1000 Hz and considering the output as measured in the intraarterial fluid to be 100%. Then, by changing only the frequency setting, we determined the speaker output for

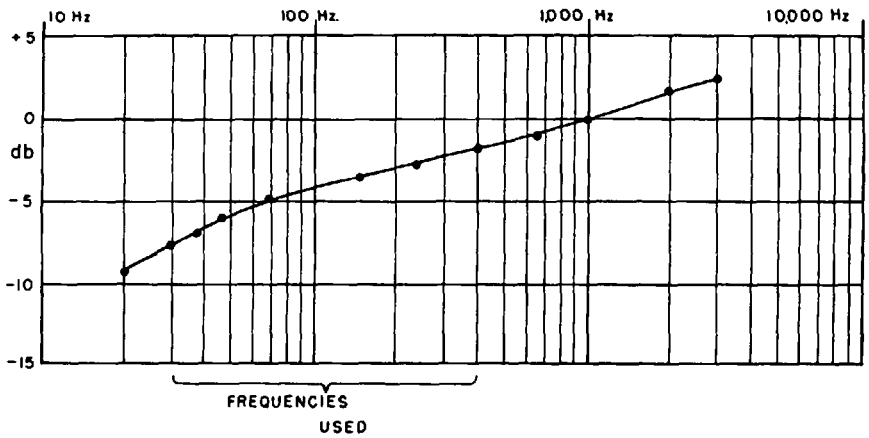

FIGURE 3

Loudspeaker output with 100 mm Hg pressure in the tubing leading to the vessel.

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frequencies above and below 1000 Hz. As shown, the amplitude decreased as the frequency decreased and increased as the frequency was raised.

TRANSMISSION OF VIBRATION

The measurements of the amplitude and frequency of the vibration within the intraarterial fluid were made with a transducer (Stow Laboratories, Pitran Model PT-2) mounted in the wall of the plastic tubing leading from the speaker to the vessel. This transducer had a flat frequency response from 1 Hz to 150 kHz. A matching Pitran PT-2 transducer was mounted in the wall of the plethysmograph to monitor the vibrations transmitted to the fluid surrounding the artery. However, its usefulness in this regard was limited. Vibrations transmitted from the length of plastic tubing upon which the vessel was mounted and extending into the plethysmograph could not be differentiated from vibrations passing through the vessel wall.

This second transducer was utilized, however, by altering the physical setup of the apparatus (Fig. 4). Arteries were attached to the lengths of plastic tubing, filled with fluid at variable pressure, and suspended, under a slight degree of longitudinal tension, in air. They were kept from drying by applying the merthiolate solution from a dropper two or three times per minute. The second transducer was removed from the wall of the plethysmograph and suspended just above, but not touching, the arteries. A drop of fluid introduced between the artery and transducer made the contact by surface tension. The response of the arterial wall from 10 to 1000 Hz could then be recorded and compared to the intraluminal vibrations. The outputs from the matching transducers were connected to the X and Y matching amplifiers of a Dumont Model 401B oscilloscope. The relative amplitude of the frequencies inside and outside the vessel were measured. In addition, the transducer outputs were connected to a two-channel recorder (Sanborn Twin-Viso Model 60-1300). The response of this recorder was flat only to 30 Hz.

Both recorders demonstrated that the vibrations were transmitted readily through the arterial wall and were of identical contour on both sides of the vessel wall. There was no evidence of reflections within the wall despite its multilayered structure (Fig. 5).

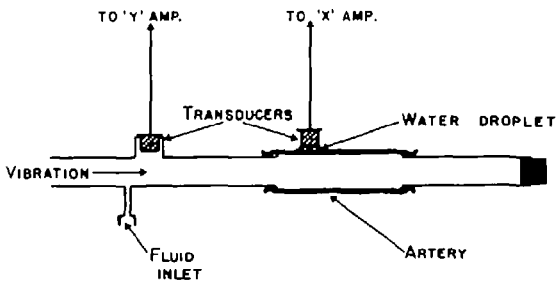

FIGURE 4

Alternate setup to measure the transmission of vibration through the arterial wall. The external transducer was coupled directly to the wall by surface tension of a drop of water. The vessel was distended at 100 mm Hg pressure and vibrations from 10 Hz to 1000 Hz were produced within it. Internal and external amplitudes of the various frequencies were compared by the display on as oscilloscope with matching X and Y amplifiers.

EFFECTS OF VIBRATION

The amplitude of the frequencies used to vibrate the vessels was selected arbitrarily since no information is available on the energy content of murmurs. At 100 Hz, when a stethoscope was applied to the apparatus, the amplitude was similar to that of a grade 3/6 murmur. The settings on the amplifier and frequency generator were not altered from this output when the different frequencies were used.

The actual vibration of the arteries took place while the vessels were distended with an intraluminal pressure of 100 mm Hg. This high pressure maintained for a period of hours caused some "creep" (a progressive extension) so that the circumference, and therefore the vessel volume, increased with time. This viscous property of the wall produced a rate of volume increase that varied from one vessel to another but usually assumed a characteristic change with time (Fig. 6). The duration of this creep was repeatedly verified and carried on much longer than expected. The rate was very rapid over the first hour, so volume measurements were usually not begun until about 4 hours after mounting. Over the succeeding 24 hours, the creep continued at a moderately rapid rate and thereafter slowed greatly but was rarely complete before 60 hours. To interpret our results, this predicted increase in volume was used as a baseline and dilation due to vibration could be shown only by a significant deviation from this line.

FREQUENCIES USED

The frequencies selected to vibrate the arteries fell within the range of frequencies reportedly contained in murmurs (15). We divided these frequencies, for the purpose of interpretation, into three groups: (a) frequencies less than 100 Hz., (b) frequencies from 100–200 Hz, (c) frequencies greater than 200 Hz.

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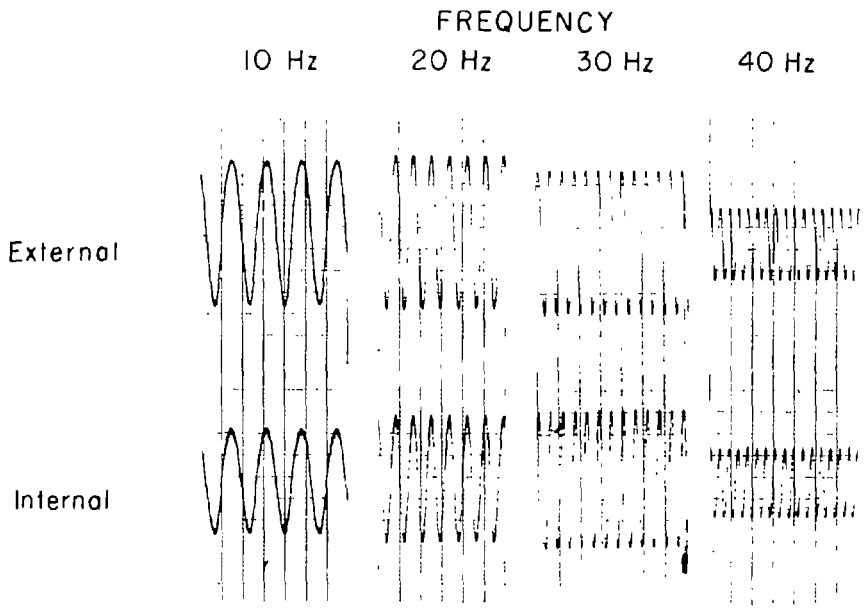

FIGURE 5

No change in the shape of the generated sine wave was noted on passage of the vibrations through the vessel wall. Recordings illustrated here were taken on the Sanborn Twin-Viso recorder and amplitude settings are random. A slight phase lag is seen due to the distance between the transducers.

Results

DILATION

Twenty-four human external iliac arteries were vibrated and 19 dilated in response to one or more of the frequencies to which they were exposed. The rate of dilation was exceedingly slow but could be measured over a period of 7 to 10 hours. The percent increase in radius, in excess of that expected for creep and projected for 24 hours, was 7.1 ± 3.4 (SD) % per day. In a series of 26 canine carotid and femoral arteries banded by Roach (5), maximal dilation was seen by 10 days, and the total increase in diameter varied from 5% to 25%. Therefore, our results showed somewhat more rapid dilation but are still comparable to that found in vivo in dogs.

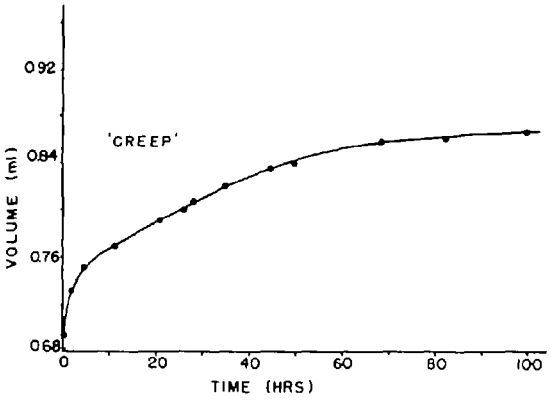

FIGURE 6

A gradual increase in the vessel volume (or radius) occurred when the vessels were held at 100 mm Hg. This is creep, a viscous property of the vessel wall. The general shape of this curve was consistent from vessel to vessel with an initial rapid increase in volume followed by a very slow distension.

VARIABILITY OF RESPONSE

It was soon apparent that any one artery did not dilate with every frequency to which it was exposed. An artery which failed to dilate when exposed to one frequency might very well dilate when exposed to another (Fig. 7). Several variables were considered in attempting to decide what factors governed the frequency response of an artery.

The first consideration was the *energy* being delivered to the arterial wall. At the higher

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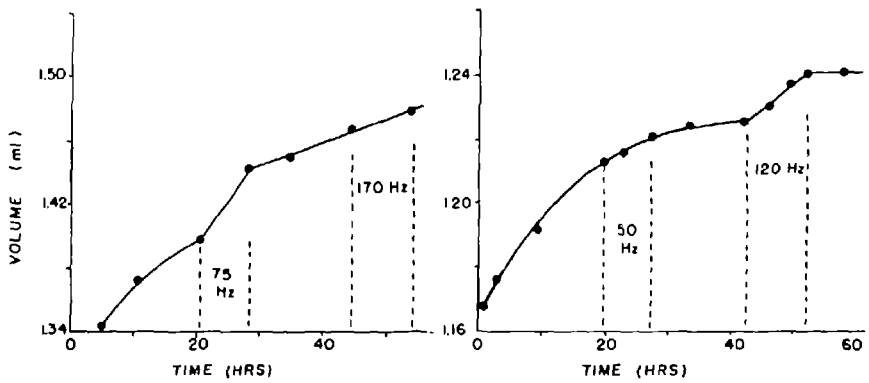

FIGURE 7

Left: *Segment of external iliac artery from a 33-year old male showed dilation, in excess of that expected from creep, when exposed to 75 Hz but not when exposed to 170 Hz.* **Right:** *Segment of external iliac artery from a 52-year old male did not dilate with 50 Hz but did with 120 Hz.*

frequencies, this energy was much greater than that being delivered at the lower frequencies ($E \alpha f^2A^2$, where E = energy, f = frequency, and A = amplitude). The lower amplitude being produced by the speaker at the lower frequencies made this difference even greater. However, despite this energy difference, it was not uncommon for an artery to dilate in response to a low frequency and not to a higher frequency. The reverse was also seen, and it was therefore concluded that energy content was not the sole factor.

A second possibility was that the sound, or an overtone of it, whose wavelength corresponded to the length of the segment of vessel in use, was setting up a *resonance* peak resulting in the dilation. Wavelength depends directly on the velocity of sound in the fluid medium and inversely as the frequency ($v = f\lambda$, where v = velocity, f = frequency, and λ = wavelength). The velocity of sound in arteries is about 5 m/sec (8, 16) and depends on the properties of the walls (16). The lengths of vessels used varied from 1.15 cm to 3.00 cm (mean 2.10 ± 0.50 cm), so a resonance peak could be a consideration. However, no evidence of resonance peaks was found either inside or outside any aged vessel where the outputs from the matching transducers were compared by the alternate setup (Fig. 4). Also, no attenuation of the vibration for any frequency from 5 to 1000 Hz was measurable on passage of the frequencies through the vessel wall with the vessel distended at 100 mm Hg. Lateral vibration of the entire vessel segment was unlikely because of the longitudinal tension present. A plot of the length of the vessel versus the frequency causing dilation showed no correlation.

The elastic properties of the arteries are known to change with age (17), and so a third variable, the age of the artery being used, was considered. By dividing the vessels into three groups—6 vessels less than 45 years, 11 vessels 45–60 years and 7 vessels over 60 years old, a correlation appeared between frequency, age, and dilation (Table 1). Statistical analysis revealed the frequency response between the youngest and oldest age groups to be significantly different ($\chi^2 < 0.05$). The middle-aged group, although appearing to respond mainly to the middle range of frequencies was not significantly different from either of the other two groups ($\chi^2 > 0.20$). However, from these results we have concluded that the older the artery the higher is the frequency of sound to which it will likely respond with dilation.

ALTERATIONS IN ELASTIC PROPERTIES

In all cases, when dilation occurred, the tension-radius curve was shifted to the right, but the final slope, which represents the elastic

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TABLE 1

Response of Arteries for Various Frequency and Age Groups

Frequency	Age (yr)	Dilated	No dilation
<100 Hz	16–45	5	1
	45–60	2	6
	60+	1	5
100–200 Hz	16–45	1	2
	45–60	5	1
	60+	0	5
>200 Hz	16–45	0	4
	45–60	1	6
	60+	7	1

modulus of arterial collagen (12), was unchanged (Fig. 8). Vibration altered the initial part of the curve, which represents the modulus for the arterial elastin, and had therefore made some alteration in the elastin network so that it became more distensible. Thus a greater radius was achieved before the unaltered collagen fibers were stressed. These changes corresponded to those found in poststenotic dilation of arteries both in vitro (10) and in vivo (3).

HISTOLOGY

Histologic examination, by light microscopy, of dilated arteries fixed at various pressures from 35 to 100 mm Hg and stained with Weigert's stain revealed no consistent changes compared with the controls.

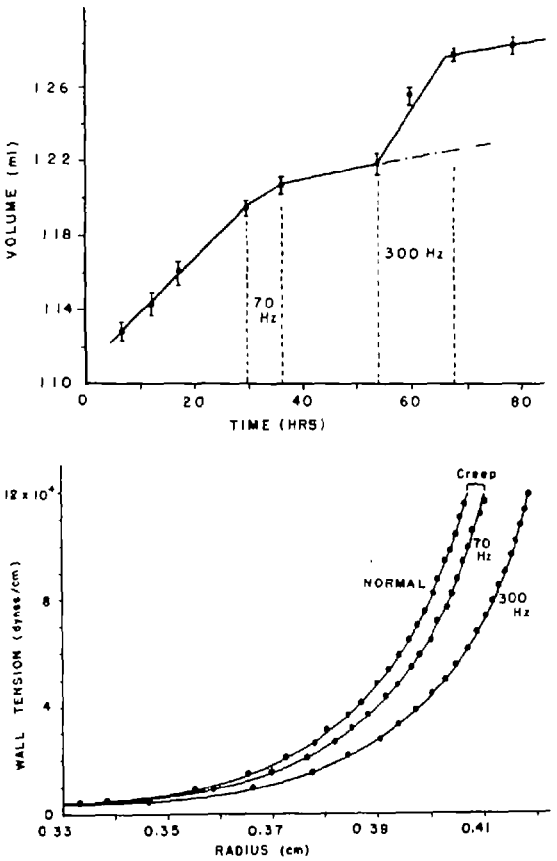

FIGURE 8

Changes in the elastic diagram with creep were identical to those produced by vibration. **Top:** *72-year old artery dilated with 300 Hz but not with 70 Hz.* **Bottom:** *Shift of the tension-radius curve to the right with 70 Hz is due only to creep but the shift with 300 Hz is greater than would be expected from creep alone.*

Discussion

Wolinsky and Glagov (18) studied the structure and function of the various components of rabbit aortic media at various distending pressures. They demonstrated that below physiologic pressures the elastin lamellae are gradually stretched and the interlamellar spaces gradually decrease. Connecting these lamellae are many fine elastin nets which, as the tension increases in the wall, come to lie circumferentially and apparently act to distribute the stress throughout the wall. Collagen fibers, which lie between the lamellae, are randomly oriented until about 80 mm Hg pressure and by 120 mm Hg pressure all are oriented circumferentially. These histological findings, therefore, correspond well to the two-component tension-radius diagram, with elastin providing the tension at low pressures and collagen at high pressures (12).

Since the effects of vibration did not alter the final slope of the elastic diagram, it is unlikely that any change in the collagen had occurred. The damage appeared to be to the elastin network. If this is the case, the damage is not likely to be found in the elastin lamellae for two reasons. First, changes here should be readily visible on light microscopy, and second, if the lamellae were broken up, the elastic diagram would no longer be a two-

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component curve. The collagen alone would be left to produce the wall tension, and since it has a high elastic modulus the curve would turn upward rapidly (i.e., be shifted to the left). This was not the case.

The most likely explanation for the findings is that changes were produced within the elastin nets joining the lamellae. Breakdown of these nets would allow an increase in the overall distensibility of the arterial wall when elastin provides the wall tension. The longitudinal transmission of vibration, "shear waves" (19), is the most likely cause of tissue disruption and could not be assessed by our apparatus (Fig. 4).

The reason for the apparent change with this important band of frequencies with age can also be suggested. Roach and Burton (17) showed that the elastic diagram for the arterial wall shifts to the left with increasing age. The changes that occur in the wall appear to influence mainly the initial portion of the curve, where a decreased distensibility is seen, and changes in the nature of the elastin likely account for this increased stiffness. Hass (20) demonstrated that isolated aortic elastin shows decreasing distensibility with age, and fluorescent spectra suggest increased elastin crosslinking (21). Such changes could account for the alteration of the frequencies necessary for elastin disruption.

The elastin nets may also be responsible for the "creep" phenomenon observed and used as a baseline. A material which normally obeys Hooke's law as a single fiber, when woven into the form of a net, can display viscoelastic properties. Not only will a hysteresis loop be produced, but "creep" can be demonstrated (22). Remington (23) demonstrated that the hysteresis loop is not a specific characteristic of muscle alone but can be seen with elastin, e.g., ligamentum nuchae. Creep and stress relaxation were also demonstrated for the ligamentum nuchae but were not present for tendon (collagen). Apter (24) demonstrated viscoelastic properties for aortic elastin, but aortic collagen showed only elastic properties.

The possibility that disruption of muscle fibers was responsible for the changes observed following vibration seems unlikely. Dobrin and Rovick (25) showed that vascular smooth muscle contraction produces an increased elastic modulus at all but the highest stresses. By exposing vessels first to norepinephrine to produce muscle contraction then to potassium cyanide (KCN) to remove all muscular activity, they illustrated the differences in the elastic diagram produced by smooth muscle. From their results we would expect the breakdown of muscle fibers in spasm to make the vessel more distensible at both high and low stress. Such was not the case. The increased distensibility we found after vibration was limited to the lower values of wall stress, and our tension-length diagrams most closely resembled the vessels exposed to KCN.

The question of resonance of the arterial wall in response to vibration remains unanswered. We found no evidence of resonance peaks of the type reported by Foreman and Hutchison (11) for the arterial wall, and there was no apparent attenuation of any frequency over the range tested. It is possible that we may have missed such peaks if they were small or of a narrow frequency band but Lees and Dewey (26) found no evidence of resonance peaks in four murmurs recorded from human carotid and femoral vessels in vivo.

Since Bergel (14) showed that, in dogs, the static mechanical properties of femoral and carotid arteries, as well as abdominal and thoracic arteries were similar, we feel that our findings in human external iliac arteries can be projected to other large vessels in the body. To verify this assumption, one carotid artery, age 68, was obtained and exposed to various frequencies. The vessel failed to dilate to the low (70 Hz) and middle range (160 Hz) frequencies but did dilate to a vibration of 325 Hz.

Thus a tentative explanation can be offered of why some patients show marked poststenotic dilation while others may show minimal or occasionally no dilation. If the murmur being produced by the stenosis contains no frequency components likely to give dilation in that

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age group, none will occur. Also, if a murmur has predominant frequency components in the age group's susceptible range, marked dilation can occur.

In addition, these experimental findings may offer an explanation for pulmonary artery dilation observed with atrial septal defect and other left-to-right shunts. A "flow murmur" occurs within the pulmonary artery when the flow rate exceeds the critical Reynolds number and turbulence is produced. This murmur then transmits its vibrational energy to the arterial wall and dilation develops in the same way as it does distal to a stenosis (27).

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