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FINAL REPORT

FLUID DYNAMIC ASPECTS OF CARDIOVASCULAR BEHAVIOR
DURING LOW-FREQUENCY WHOLE-BODY VIBRATION

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Astronautical Engineering

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REPORT

By

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On.................................FLUID DYNAMIC ASPECTS OF CARDIOVASCULAR................

....BEHAVIOR DURING LOW-FREQUENCY WHOLE-BODY.............

....VIBRATION

For the period.......1 November 1968 - 31 October 1972

Submitted by.........Dr. Robert M. Nerem.........

.........Department of Aeronautical and Astronautical
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ABSTRACT

The behavior of the cardiovascular system during low-frequency whole-body vibration, such as encountered by astronauts during launch and re-entry, is examined from a fluid mechanical viewpoint. The vibration characteristics of typical manned spacecraft and other vibration environments are discussed, and existing results from in vivo studies of the hemodynamic aspects of this problem are reviewed. Recent theoretical solutions to related fluid mechanical problems are then used in the interpretation of these results and in discussing areas of future work. Included are the results of studies of the effects of vibration on the work done by the heart and on pulsatile flow in blood vessels. It is shown that important changes in pulse velocity, the instantaneous velocity profile, mass flow rate, and wall shear stress may occur in a pulsatile flow due to the presence of vibration. The significance of this in terms of changes in peripheral vascular resistance and possible damage to the endothelium of blood vessels is discussed.

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I. INTRODUCTION

Interest in the effects of low-frequency whole-body vibration on the behavior of the cardiovascular system stems from many possible applications. These include:

(i) the effect of spacecraft vibrations on astronauts during launch and re-entry and possible similar effects for others who must operate in a vibration environment, e.g., jackhammer operators, helicopter crew members and passengers, etc.;

(ii) the use of vibrations as a cardiac-assist device because of the decreased peripheral vascular resistance and blood pressure levels and the increased cardiac output -- in the Netherlands a technique of this general type is being used to bring people out of cardiogenic shock [1];

(iii) the use of vibration as a controlled means of perturbing the cardiovascular system and thus as a diagnostic tool;

(iv) the use of vibrations as a means of producing an artificial gravity effect in space vehicles [2].

Many of the effects of vibration are obviously of a complex physiological nature. However, within these there appear to be fluid mechanical aspects of the problem which need to be investigated. It has thus been the purpose of this research effort to investigate the whole-body vibration problem from a fluid mechanical viewpoint. Following a brief review of the type of vibration environments in which man operates and of past in vivo studies of cardiovascular behavior during vibration, three specific problems in which the fluid mechanics may be an important aspect and which have been considered as part of this effort will be discussed. These problems are: (i) the direct effect of vibrations on the pressure distribution in the heart and on cardiac work; (ii) the influence of vibration on the nature of arterial pulse wave propagation; and (iii) the effect of whole-body vibrations on flow in the vascular bed and of vibration-induced stresses on the endothelium.

II. MAN VIBRATION ENVIRONMENTS

Figure 1 shows the kind of vibration amplitudes to which man may be exposed during whole body vibration at low frequencies. Included are the characteristics of early pneumatic tools [3], Janeway's standard of the automotive industry [4], the WADC vibration tolerance
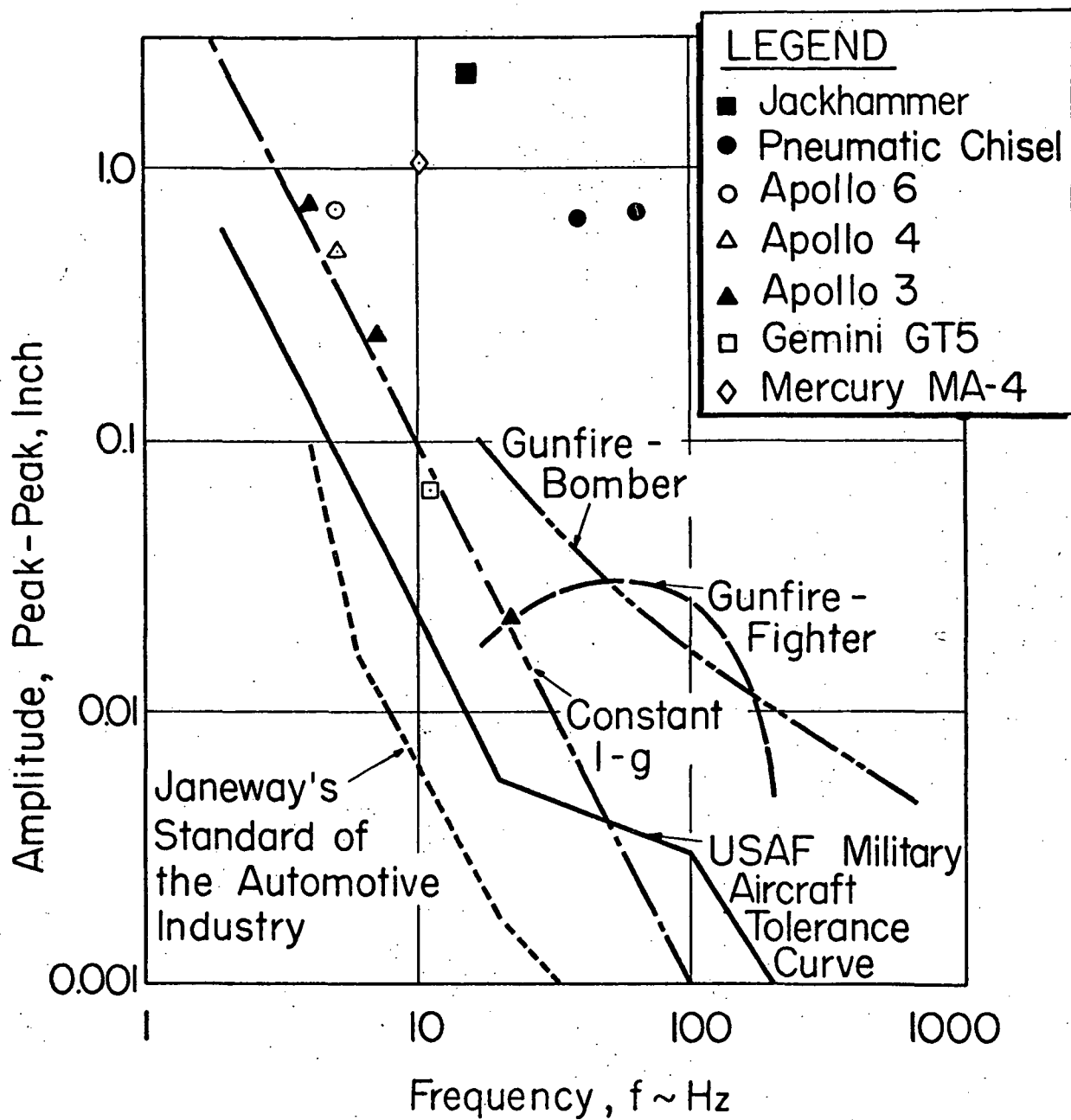
Fig. 1 - Low-Frequency Mechanical Vibration Environments

curve and typical vibration environments associated with 0.50-caliber machine gun fire [4], and typical spacecraft vibration data obtained in the manned space flight program of the United States [5]. Also shown is a line corresponding to a 1 g peak-to-peak acceleration; and as may be seen, vibration accelerations on that order or even greater are encountered in some man-operating environments.

Historically, the interest in whole-body vibration effects goes back at least 50 years to the work in Rome by Loriga [6] who was interested in injuries to workers who used pneumatic tools for long periods of time. Studies of such effects have continued over the years with primary interest being focused on the circulatory disease known as "Raynaud's phenomenon of occupational origin" [7]. Symptoms of this disease include numbness and clumsiness of the fingers and intermittent blanching of the hands; and although there are many factors which appear to make up these symptoms, exposure to vibration has been shown to be an important ingredient with the incidence of Raynaud's phenomenon increasing with increased exposure to vibration. All evidence does indicate, however, that attacks continue to persist even though the worker is removed from the vibration environment with which the symptoms were first associated.

More recently, investigations of vibration effects have been oriented towards the problems encountered in aircraft and spacecraft. The results of related in vivo studies will be briefly reviewed in the following section; however, it is noted that one of the earliest fluid mechanical studies of the effects of vibration [8] was motivated by reports of possible fuel starvation of jet aircraft engines during the firing of machine guns. Lance concluded that it was "possible for the flow of a viscous fluid, in a pipe or channel, to be arrested when the pipe is subjected to one or more pulses of sufficient strength" and determined the necessary conditions for this to occur. Certainly, if such effects are possible in the constant pressure-gradient flow analyzed by Lance, then similar effects as discussed in Section VI could occur in the vascular bed.

Vibration problems have also been encountered during launch in the Mercury, Gemini, and Apollo programs [5]. These are depicted in Figure 1; and in the latter case range from a buffeting type of oscillation encountered on the early unmanned Apollo flights, e.g., Apollos 3 and 4, to the "pogo-type" longitudinal oscillation encountered on Apollo 6. Although on Apollo 8 and subsequent flights vibration levels were kept within a tolerance level of ± 0.25 g, it was at least in some cases at the expense of flight performance since the S-2 stage center engine was shut down early. Future space flight related interests in whole-body vibration will also include the possible use of low-amplitude vibrations to provide a type of artificial gravity.

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III. CLINICAL STUDIES

At Ohio State University the Department of Preventive Medicine has been engaged in a continuing study of cardiovascular vibration effects for the last ten years. Laboratory studies have been carried out on rats, dogs, and humans; and as a result of these experiments, a number of general conclusions have been reached with regard to low-frequency vibration effects. These conclusions are summarized in Table I and are based on the measurements in a dog obtained primarily at 4, 7, and 11 Hz and with half-amplitudes on the order of 1/4-inch [9]. As may be seen, one of the effects of vibration is a decrease in the peripheral resistance. This may be due to the dilation of the resistance vessels; however, it could equally well be due to a change in the nature of the flow, e.g., the velocity profiles in these vessels, due to a coupling of the vibrational motion with the pulsatile output of the heart. Such a coupling can actually produce a resonance-type effect with the two critical frequencies being that of the heart beat and that of the mechanical vibration.

Table I - Effects of Low Frequency Vibration on Cardiovascular Behavior

Property	Effect
Mean Systemic Arterial Pressure	Decreased
End Diastolic Pressure	Decreased
Cardiac Output	Increased
Heart Rate	Increased
$\left(\frac{dp}{dt}\right)_{MAX}$ Left Ventricle	Increased
Peripheral Vascular Resistance	Decreased

Another interesting aspect of cardiovascular behavior during vibration is the possible change in pulse wave velocity. Preliminary pulse wave velocity data have been reported [9]. These were based on measurements carried out using a vibration-compensated pressure transducer [10] to measure the shape and magnitude of the pressure pulse wave at separate locations along the aorta of an anesthetized dog undergoing longitudinal vibration. Although only a limited number of measurements were obtained, these indicate a marked increase in the pulse wave velocity at a frequency of 7 Hz. This result will be discussed further in Section V.

Studies of whole-body vibration effects have also been in process at many other research laboratories, including the U. S. Army Medical Research Laboratory and the NASA Ames Research Center. From a hemodynamic viewpoint those of most interest are the vibration studies

carried out at the Aerospace Medical Research Laboratories at Wright-Patterson AFB [11], and the previously referred to cardiac-assist experiments in the Netherlands [1]. Clark and his co-workers at Wright-Patterson Air Force Base carried out measurements of cardiac output, blood pressure, and peripheral resistance, and reached similar conclusions to those of the Ohio State studies. On the other hand, Arntzenius and his colleagues in the Netherlands have carried out experiments using piglets which demonstrate that the use of vibrations synchronized with the heart beat is an effective cardiac-assist device for treatment of cardiogenic shock as well as for use after a coronary occlusion. It is because of such demonstrated hemodynamic aspects to the cardiovascular vibration problem that the studies to be discussed in the remainder of this report were undertaken.

IV. CARDIAC BEHAVIOR

One aspect of cardiovascular behavior during whole-body vibration which is of fluid mechanical interest is the direct influence of vibration on the heart itself. This was the first major area of investigation undertaken on this Grant, and a cursory examination of this problem is afforded by using the pulsating bulb model of the heart suggested by Jones [12]. Viscous effects are negligible in the heart, with the exception, of course, of the boundary layer region on the wall of the heart; and following Jones, the velocity potential for the inviscid flow in this simple model of the heart is that of stagnation flow.

Assuming that vibration does not change the volume time-history of the heart's pumping action, then, as suggested by Howe of NASA [13], a new velocity potential is obtained through a simple coordinate transformation. The pressure variations in the heart are found using the unsteady form of Bernoulli's equation; for the case where the vibrations are along the axis of the bulb and in the direction in which fluid is expelled into the aorta, the following theoretical equation for the change in the pressure in the heart is found:

$$\Delta p_{vib} = \Delta p_{nvib} - \rho U_1^2 \bar{x}_0 (\bar{x}' - 1) f_2'' \tag{1}$$

Here p_{vib} is the pressure difference between a point denoted by $\bar{x}'$ in the heart and the entrance point to the aorta for the case of vibration, p_{nvib} is the same pressure difference for the case of no vibration, $\bar{x}_0 = x_0/L$ where x_0 is the maximum displacement during vibration and L is the distance from the center "stagnation point" of the heart to the entrance of the aorta and represents the characteristic length of the heart, ρ is the density of blood, and U_1 is the maximum cardiac ejection velocity for the case of no vibration.

f_2'' is the nondimensional instantaneous acceleration felt by the heart. The actual acceleration is $(U_1 \bar{x}_0 f_2'')/L$, and it can thus be seen that the change in pressure in the heart is directly related to the acceleration to which it is exposed. In fact, since the volume time-history of the heart has been assumed to be unchanged by vibration, then any change in pressure in the heart must simply represent a super-position of acceleration effects on top of that pressure gradient required to expel the fixed amount of fluid from the heart. For typical heart conditions and a 1 g acceleration, this pressure change is at the most of the order of 10 mmHg; it thus represents a 10 percent effect compared to the normal heart pressure level. Of course, for higher accelerations the pressure change would be greater. In addition, the cardiac output will not remain fixed, but in general will increase as is indicated in Table I.

A more complete theoretical investigation of cardiac behavior during whole-body vibration has recently been completed by Hooks [14]. In his analysis, Hooks modeled the heart as a spherical cavity with a sinusoidally varying radius and a single opening for entrance and exhaust of fluid. The damping characteristics of the body were included using a linear model with the heart connected by a spring and dashpot to the rib cage. The varying mass of the heart was accounted for and the thoracic cavity was modeled as a cylinder of fluid within which the heart is positioned.

The results of Hooks for vibration in the direction of the spine and thus parallel to the flow up through the ascending aorta are shown in Figure 2. Here the mechanical pumping power of the heart is presented as a function of the heart vibration frequency. These calculations have been carried out for varying phase angles; both the maximum and minimum work rates are shown, as well as the average pumping power obtained by averaging over phase angle, all for a fixed cardiac output and aortic pressure time-history. For the case where the heart and vibration frequencies are not equal, this average pumping power is representative of the long-term vibration effect; and, as may be seen, there is no significant effect. However, for the case of the two frequencies being equal, the phase relationship between the two remains constant; the heart may operate anywhere between its peak and minimum power requirements depending on the phasing of the vibration. The magnitude of this effect is seen from Figure 2 to be in the range of less than 10 percent. It should be noted, however, that the major uncertainty in the calculation is the pressure gradient in the thoracic cavity and the influence of this external pressure on the pumping heart. The model of Hooks tends to maximize this effect and thus the actual change in the pumping power of the heart may be much less.

It should also be noted that the heart pressures in equation (1) were referenced to the aortic pressure since it is that difference which results in flow out of the heart. However, the aortic pressure itself may change due to an influence of vibration on the resistance of the
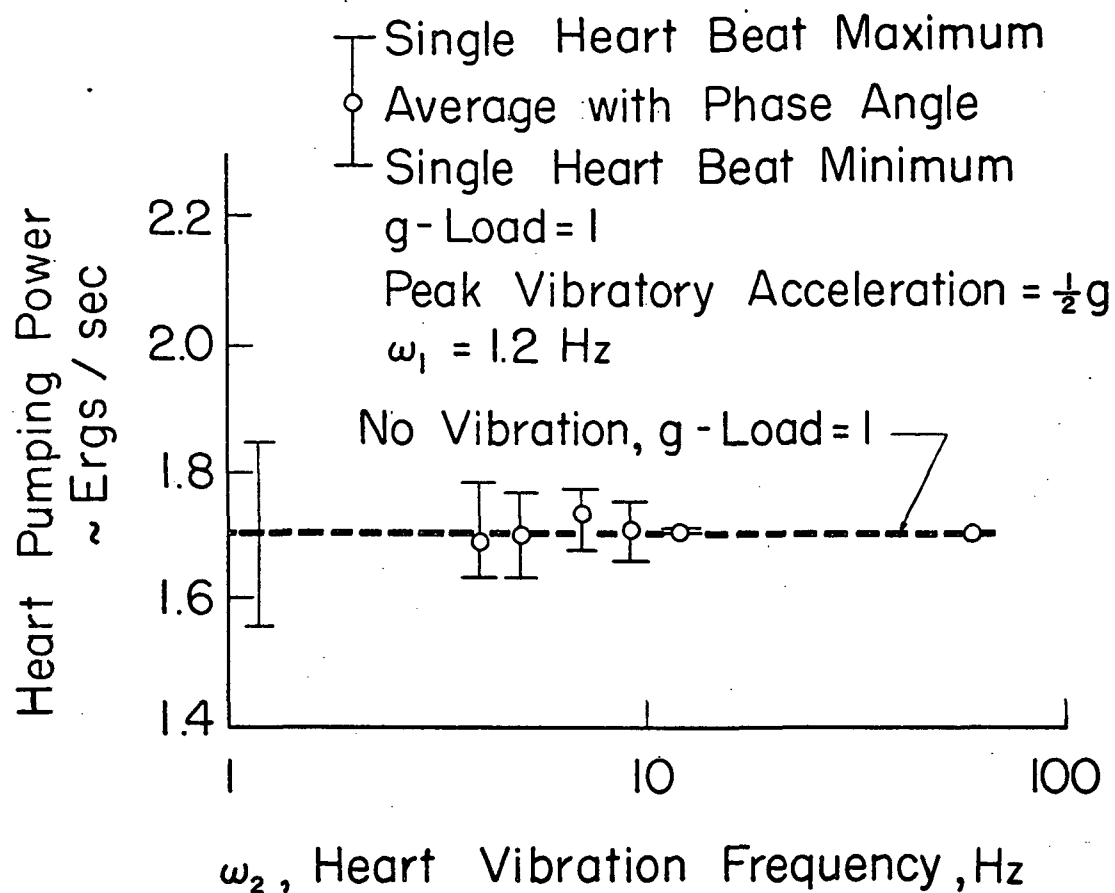
Fig. 2 - Mechanical Pumping Power of the Heart During Vibration
Parallel to Ascending Aorta

vascular bed. Thus, while the direct influence of vibrations on the heart may appear to be small, based on the results presented here, an indirect vibration effect associated with change in peripheral vascular resistance may, in fact, be large. This is certainly suggested by the laboratory observations reviewed in Section III, and some of the fluid mechanical aspects of this will be discussed in Section VI.

V. PULSE WAVE PROPAGATION

Previous investigators have noted a change in pulse velocity due to the influence of whole-body vibration [9]. Whether this is a real change, e.g., due to a possible influence of vibration on the nature of the arterial wall properties, or an apparent change, e.g., due to a change in the characteristics of the arterial pulse pressure waveform, is not clear.

Thus, in order to investigate this further, blood pressure waveforms have been monitored in the upper descending aorta of the dog, and their characteristics analyzed [15]. The measurements were obtained using two acceleration-compensated pressure transducers mounted 10 cm apart on a flexible catheter [10]. The animals were secured in a supine position on a shake table and were vibrated sinusoidally along the longitudinal body axis at frequencies ranging from 2 to 16 Hz and with a 1/2-inch double amplitude.

The data obtained suggests that the arterial pressure wave in the presence of vibration is a superposition of a sinusoidal pressure wave, whose frequency corresponds to the table vibration frequency, upon the natural aortic pressure pulse. The effects of this sinusoidal wave, which is excited by the longitudinal vibrations, becomes evident from comparison of results of Fourier analysis of the pressure pulses measured for vibration and nonvibration conditions. Calculations of the apparent phase velocity of each harmonic, i.e., the spatial rate of change of phase of the harmonic, were made. These calculations revealed a significant change in the apparent phase velocity of those harmonics whose frequency corresponded to the table vibration frequency (and hence, the frequency of the sinusoidal wave). This is illustrated in Figure 3 where V.F./H.R. is the vibration frequency-heart rate ratio.

These effects on apparent phase velocity were most pronounced when the critical harmonics were of harmonic number 4 and above. The magnitude of the critical harmonic was generally observed to be increased from the corresponding averaged control value. The apparent phase velocity of the critical harmonic was observed to assume a wide range of values for the pulses recorded during vibration, however a majority of these values showed significant increases or decreases from corresponding averaged control values. Often the apparent phase velocity of
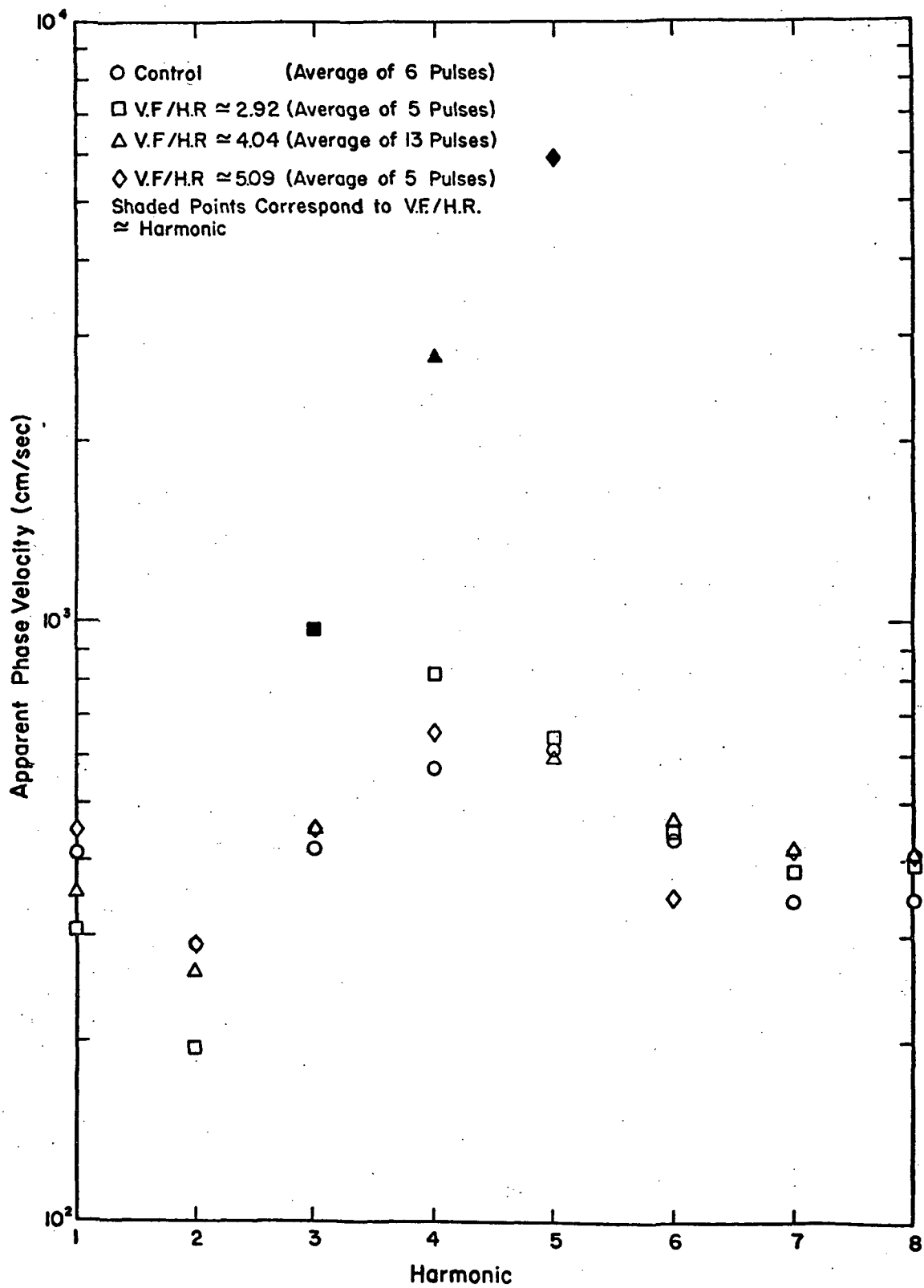
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Fig. 3 - Apparent Phase Velocity as a Function of Arterial Pressure
Harmonic Number

the critical harmonic assumed negative values as well. Harmonics other than the critical one showed little variation in magnitude from the corresponding averaged control values, but did show changes in apparent phase velocity for random harmonics of a limited number of pulses. Such deviations may be attributed in part to the effects of the vibration-induced wave. Since the vibration-induced wave is not purely sinusoidal and thus has its own harmonic content, it may interact with several of the pulse wave harmonics. Furthermore, nonlinearities which are present in the arterial system and which are neglected in this analysis, would account for increased interaction of the harmonic components of the pulse wave.

A linearized theory of wave propagation [16] through an elastic tube which contains an incompressible, viscous fluid and which is longitudinally tethered and surrounded by a viscous medium of finite mass was utilized to investigate theoretically the observed characteristics of the vibration-induced wave. This theory predicts the possible presence of two types of waves of markedly different phase velocity and damping characteristics for a fluid-vessel system undergoing axisymmetric motion, as is assumed here. The type I wave has been shown to be primarily associated with radial motion of the vessel wall, while the type II wave is principally associated with longitudinal motion of the vessel wall.

Using the superposition principle which is applicable to a linear system, it was assumed that the vibration-induced wave and the critical harmonic component of a control pulse wave are sine waves which are linearly combined, resulting in a third sine wave representing the critical harmonic component for a corresponding vibration pulse. It was then possible to calculate magnitudes and apparent phase velocities of the resultant wave by either assuming the vibration-induced wave to be a type I or a type II wave. Such calculations, which were highly dependent upon the relative magnitudes and phases of the component waves, yielded an extensive range of possible values for the magnitude and apparent phase velocity of the critical harmonic. These calculations, both for the case in which the vibration-induced wave is assumed to be a type I wave and for the case where it is assumed to be a type II wave, predicted critical harmonic magnitudes and apparent phase velocities that were observed experimentally. However, comparisons of the theoretical calculations with the experimental measurements for these two variables were not successful in substantiating the hypothesis that the vibration-induced pressure wave is a longitudinal type II wave. Thus, unfortunately the exact nature of this wave could not be ascertained from the results of the present analysis.

VI. THE VASCULAR BED

In addition to the direct effect of vibrations on the heart as discussed in Section IV, whole-body vibration can also affect the heart indirectly through changes in the systemic circulation, e.g., by influencing the peripheral resistance. It is thus of interest to investigate the effect of vibrations on the flow in blood vessels. Within the cardiovascular system, there are blood vessels going in all different directions; and thus when the body is exposed to whole-body vibration in a particular direction, there is no uniformity among the blood vessels in terms of their orientation to the vibration. In this section, however, the consideration of vibration effects will be limited to longitudinal vibration, i.e., vibration in the direction of the axis of the vessel. This not only would appear to be the case where a maximum effect would be obtainable, but obviously also is one which lends itself to somewhat simpler solution because of the symmetry of the flow.

Considering first the larger vessels, e.g., the aorta and larger arteries, the flow there is primarily inviscid with a thin boundary layer on the wall of the tube whose thickness is dependent on the unsteadiness parameter, $\alpha^2 = (\omega_1 R^2)/\nu$, where ω_1 is the fundamental frequency of the pulsatile flow output of the heart. The effect of longitudinal vibration would in these larger vessels be limited to this thin boundary layer and can be analyzed using a modification of Stokes second problem [5].

Considering parallel, incompressible flow, the governing axial direction momentum equation is

$$\frac{\partial u}{\partial t} = -\frac{1}{\rho}\frac{dp}{dx} + \nu \frac{\partial^2 u}{\partial y^2} \tag{2}$$

For a sinusoidal pressure gradient, dp/dx, and representing the vibration by a sinusoidal rigid wall motion of the form $x_w = -x_0 \sin(\omega_2 t + \varphi)$, the resulting solution for the velocity profile is [17]

$$u = U_1 \cos \omega_1 t - U_1 e^{-k_1 y} \cos(\omega_1 t - k_1 y)$$

$$- x_0 \omega_2 e^{-k_2 y} \cos(\omega_2 t - \varphi - k_2 y) \tag{3}$$

Here U_1 is the peak velocity in the inviscid portion of the flow (note that the leading term in equation (2) is the time-variation of the inviscid flow velocity), ω_2 is the frequency of the vibration, φ the initial phase angle between the pressure gradient and the vibration, x_0 the maximum amplitude of the wall motion, $k_1^2 = \omega_1/2\nu$, and $k_2^2 = \omega_2/2\nu$.

Typical velocity profiles are shown in Figure 4 and the effect of vibration is clearly evident. However, as noted earlier, this effect
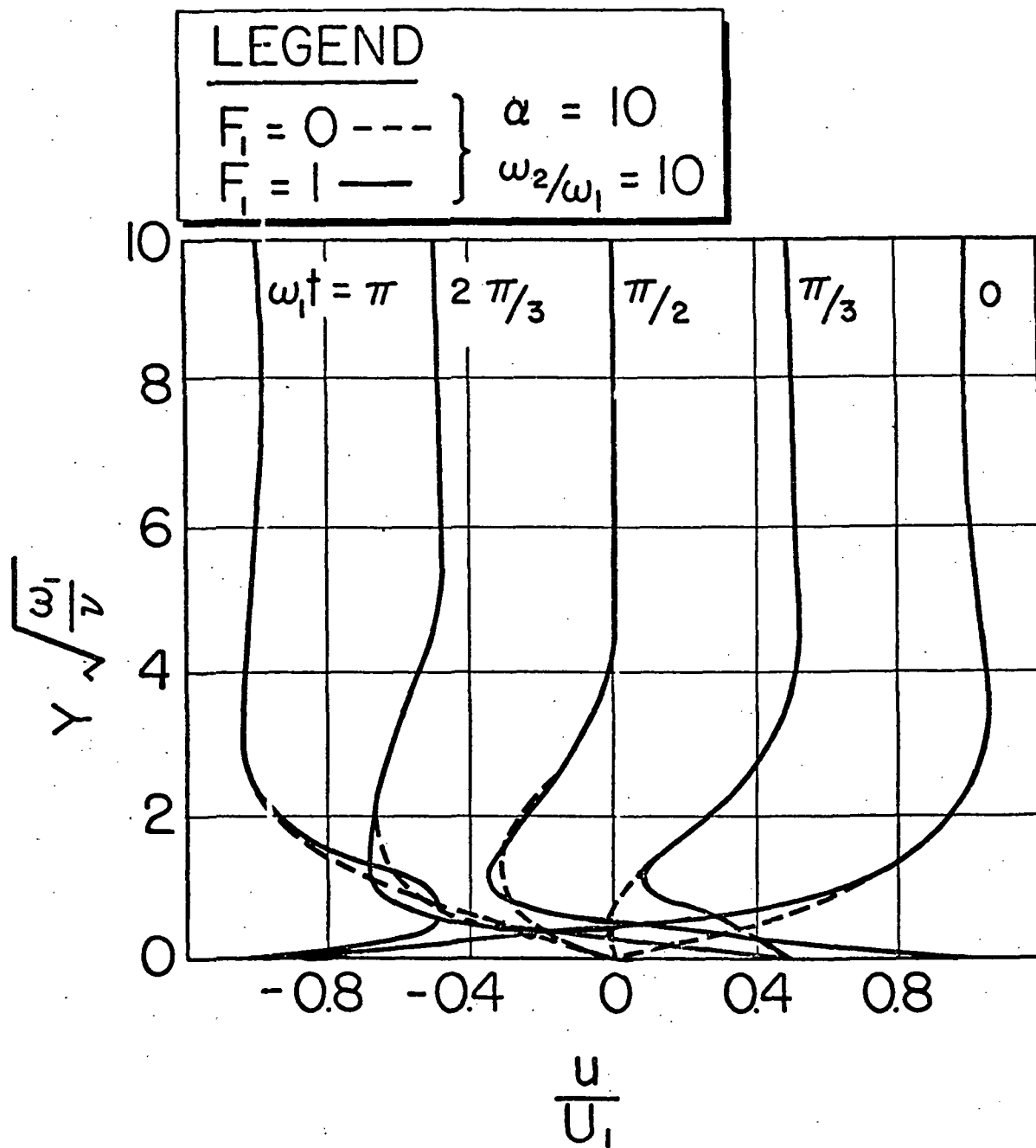
Fig. 4 - Typical Pulsatile Flow Velocity Profiles With and Without
Vibration as Calculated from Equation (3)

is limited to the thin wall boundary layer, and since the pressure drop in the aorta is small, any effect here of vibration on peripheral resistance would also be small. As also was noted earlier, there is evidence that the pulse wave velocity can be affected by whole-body vibration. However, the preceding analysis, in not accounting for the elasticity of the wall, is not able to include the effect of vibration on pulse wave velocity.

In the smaller blood vessels, the flow is fully viscous. For the case of no vibration this problem has been solved in the literature using primarily two different methods [18]: (i) the approach of Womersley [16] in which the Navier-Stokes equations and the governing differential equations for an elastic wall are linearized and then solved simultaneously yielding Bessel function solutions; and (ii) the one-dimensional flow method-of-characteristic solutions in which non-linearities may be retained. However, there is another method of solution which has found wide application in viscous flow problems, but which has until recently not been applied to pulsatile flows. This is the so-called momentum integral method, and it appeared to be a convenient method to use in investigating possible longitudinal vibration effects in a pulsatile flow. In this method, it is not the Navier-Stokes equations themselves that are solved, but the integrated form of the streamwise-direction momentum equation. The form of the velocity profiles is assumed a priori with unknown coefficients characterizing the profile, and these coefficients and the solution for the entire flow are obtained using the momentum integral equation and judiciously chosen boundary conditions. The equations which one then must solve are primarily algebraic in nature.

To further investigate the applicability of such a momentum integral method, a solution was obtained for the incompressible fully viscous, pulsatile flow in an infinitely long rigid tube in the presence of longitudinal vibration. This is actually a linear problem; however, it has served to demonstrate the effects of vibration. The details of this method of calculation will not be discussed here since they may be found in [19]; however, calculations were carried out both for the case of no vibration as well as for the case where the frequency of the pulsatile flow, ω_1, and the vibration frequency, ω_2, are equal. In the former case of no vibration, a sinusoidal pressure gradient was used, and the results obtained were found to compare favorably with those of Womersley [16].

Calculations of this type have also been carried out for the case of vibration as represented by a sinusoidal wall motion of the form $w = w_0 \cos(\omega_2 t + \varphi)$ where $w_0 = x_0 \omega_2$, x_0 is the maximum displacement of the wall motion, ω_2 the frequency, and φ an initial phase angle. In Figures 5 and 6 results are shown for $\omega_1 = \omega_2$ and for both in-phase and out-of-phase vibration, i.e., $\varphi = 0$ and $180°$. Here the maximum mass flow rate Q_{max}, divided by that for a Poiseuille flow with a pressure gradient corresponding to the maximum value of the pulsatile flow, Q_0,
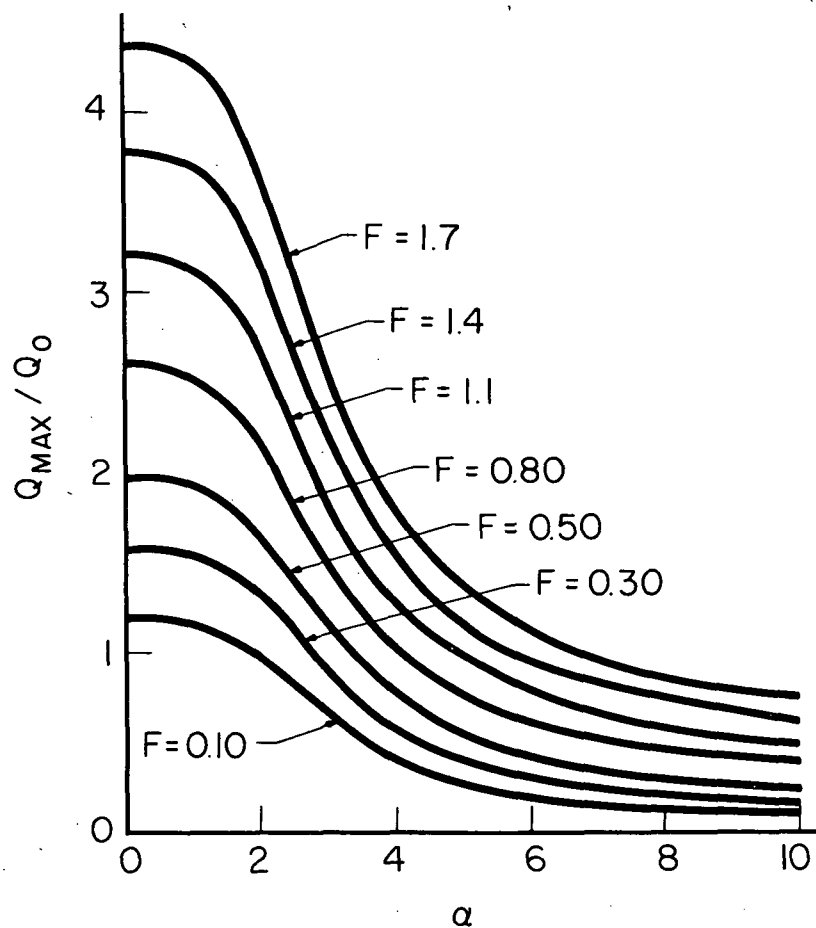
Fig. 5 - Mass Flow Magnitude in Pulsatile Flow With In-Phase
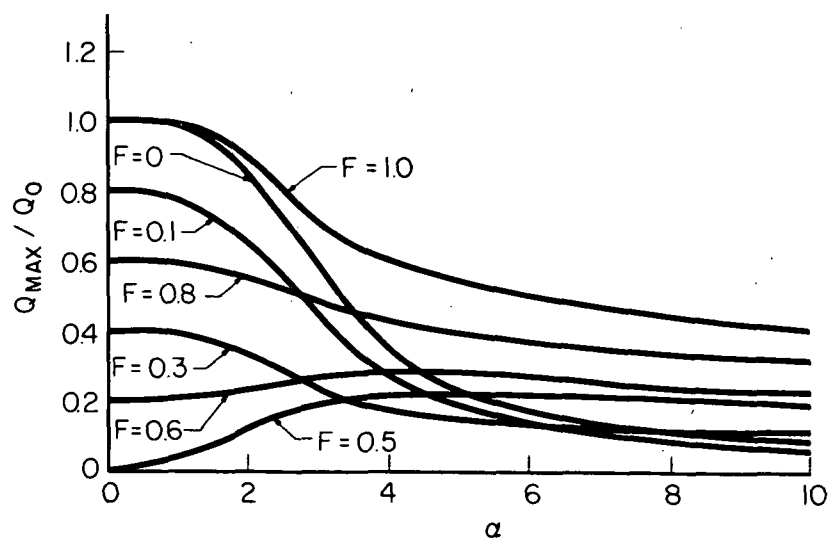Sinusoidal Pressure Gradient and Wall Motion, $\varphi = 0°$

Fig. 6 - Mass Flow Magnitude in Pulsatile Flow for Sinusoidal
Pressure Gradient and Wall Motion Acting in Opposi-
tion, $\varphi = 180°$

is shown as a function of the unsteadiness parameter, α. F is equal to $(x_0\omega_2)/U_0$ where U_0 is the centerline velocity for the corresponding Poiseuille flow with a mass flow rate Q_0. As may be seen, for in-phase vibration there is a significant increase in the maximum mass flow passed by the tube; while for out-of-phase vibration, the maximum mass flow rate is decreased for $0 < F < 0.5$ and increased, in the negative direction, for $F > 0.5$. Thus, the presence of longitudinal vibration may result in instantaneous flow stoppage and reversal. Equally important changes in the velocity profiles may also occur.

Although such instantaneous changes would appear to be important in terms of the overall vascular physiological behavior, it should be noted that, within this linear analysis, no change in the average mass flow rate over a cycle is predicted. However, the inclusion of non-linear effects would alter this; and thus the nonlinear aspects of the problem need to be investigated in the future. Whatever the exact fluid mechanical changes are, the resultant behavior must finally depend on how these aspects couple with constriction or dilation of the vascular bed.

VII. ENDOTHELIAL CHANGES

The enhancement of arterial wall shear stress by mechanical vibration has also been examined from the viewpoint of its relationship to circulatory disorders found in those using vibrating hand tools, i.e., Raynaud's phenomenon of occupational origin [20]. The point of interest is the possibility of creating wall shear stress levels sufficiently high to cause deterioration of the endothelium of blood vessels. Fry [21] has determined experimentally a critical yield stress for blood vessels of 379 dynes/cm^2, with a standard deviation of ± 22.4 percent. According to Fry, exposure to stresses in excess of this level for periods as short as one hour can result in swelling and deformation of the endothelial cells making up the inner lining. Furthermore, above an erosion stress level of approximately 1000 dynes/cm^2, dissolution of cell substances and deposition of fibrin, platelets, blood cells, and other materials on the eroded surface was found to occur.

Using the theoretical results of Section VI, the maximum wall shear stress in a pulsatile flow in the presence of longitudinal vibration may be calculated. For typical aortic flow conditions, e.g., $U_1 = 100$ cm/sec, $\nu = 0.04$ cm^2/sec, $\rho = 1$ g/cm^3, and $\omega_1 = 7.5$ sec^{-1} (72 beats/minute), then if 12 Hz vibration with an amplitude of 2.5 cm is applied the maximum wall shear stress is calculated to be in excess of 300 dynes/cm^2. For a larger vibration amplitude, the maximum shear stress will also be increased. Similar results have also been obtained using the momentum integral theory discussed in Section VI. It thus appears quite possible that shear stress levels in the aorta may exceed

the critical yield stress determined by Fry since at branching points and other similar locations even higher shear stresses may be produced than those predicted here.

The results of these calculations are summarized in Figure 7. It should be noted that these calculations do model the vibration effect as a longitudinal wall motion superimposed on the normal arterial flow. It is recognized that the amplitude of this wall motion may not correspond to that of the vibrating tool since some attenuation of the effect would be expected. Using this approach and for typical arterial flow conditions, it is estimated that shear stresses in excess of 1200 dynes/cm^2 may be produced by a vibrating tool at 20 Hz with a half-amplitude of 2.5 cm and including 50 percent attenuation of the vibration impulse. As may be seen in Figure 6, even with 90 percent attenuation of the vibration effect, the shear stress still ranges up to 300 dynes/cm^2 at a frequency of 20 Hz.

A frequency of 20 Hz and a half-amplitude of 2.5 cm is a somewhat severe vibration condition; however, there is no reason to believe, for a worker using a vibrating tool in a cold, damp environment, that the tolerance of the arterial wall to shear stresses will be the same as that for more normal conditions. Furthermore, it should be noted that in some studies of Raynaud's phenomenon [22,23], there have been reports of arterial occlusion in subjects who used some type of vibrating tool in their work. Thus, although there is no direct evidence to date that endothelial damage occurs due to vibration-enhanced wall shear stresses, the over-all evidence does suggest that there will be alterations in wall physiology and that this may be a factor in disorders induced by exposure to vibration.

VIII. CONCLUSIONS

It has been the purpose of this investigation to examine the problem of cardiovascular behavior during low-frequency whole-body vibration from a fluid mechanical viewpoint. The detailed results of this investigation have been disseminated in the publications and presentations listed in the Appendix. However, there are definite changes in pressure levels, velocity profiles, and mass flow rates which could manifest themselves as changes in peripheral resistance, cardiac output, and systemic pressure. There are also changes in arterial pulse wave propagation characteristics which manifest themselves. Finally, increases in wall shear in blood vessels due to vibration have been predicted, and it has been suggested that this may be a cause of some of the symptoms of Raynaud's phenomenon. If true, then this certainty is a long range effect from vibration which could be important for anyone operating in a vibration environment.
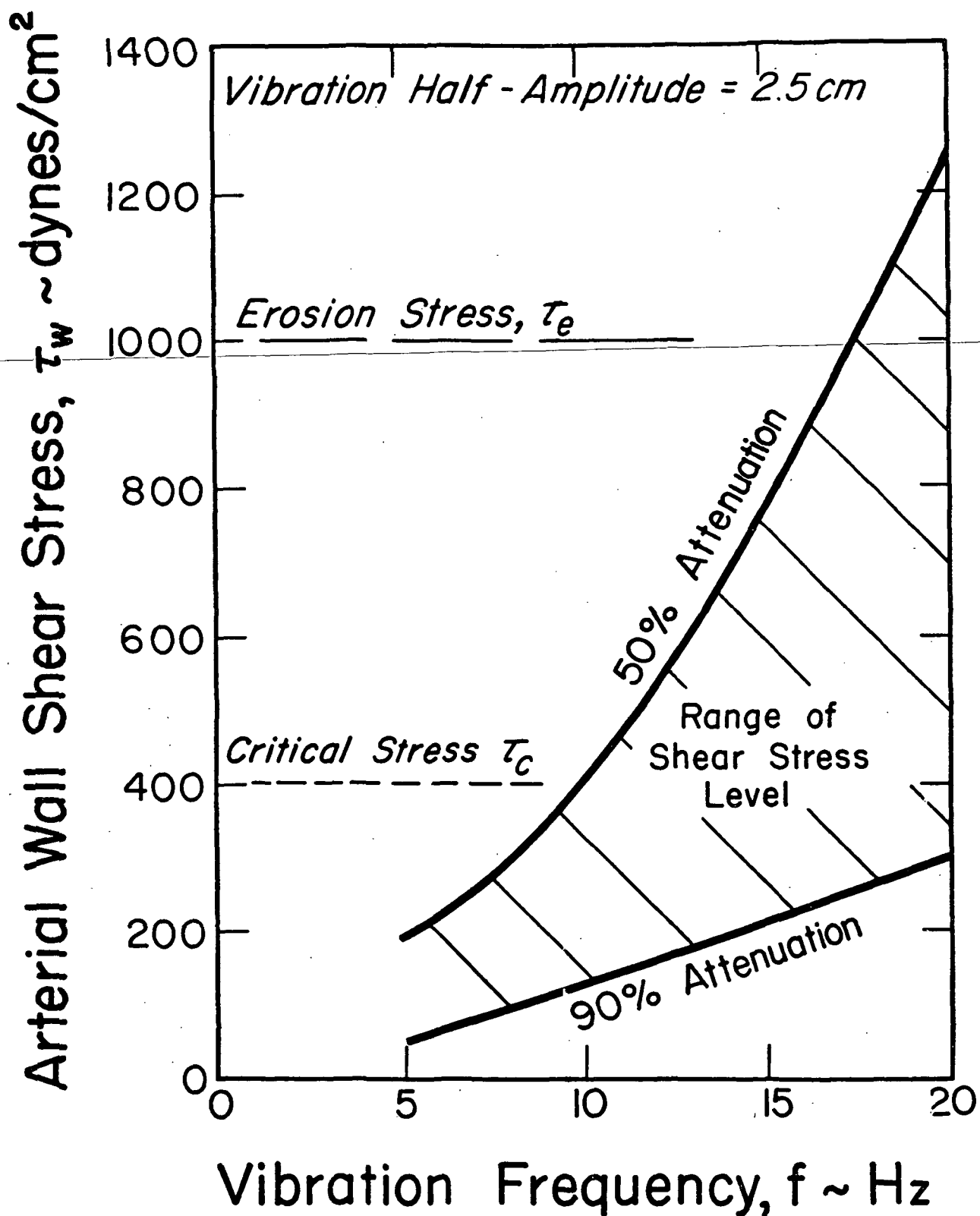
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Fig. 7 - Predicted Arterial Wall Shear Stress Due to
Longitudinal Vibration Including Attenuation
of Vibration Impulse

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REFERENCES

1. Arntzenius, A. C., Koops, J., and Hugenholtz, P. G., "Cardiovascular Responses in Piglets to Body Acceleration Given Synchronously With the Heartbeat (BASH)," presented at the 1969 American Heart Association Meeting, Dallas, Texas, November 13-16; see Supplement III to _Circulation_, Vols. XXXIX and XL, October 1969, p. III-38.

2. Gualtierotti, T., "Unidirectional Response of Stratoreceptors to Vibration - A Means for Artificial Gravity in Space Flight," presented at the 8th International Symposium on Space Technology and Science, Tokyo, Japan, August 25-30, 1969.

3. Jones, W. A., "Vibration Injury from Hand Tools - A Critical Review," M. Sc. Thesis, The Ohio State University, 1960.

4. Getline, G. L., "Vibration Tolerance Levels in Military Aircraft," presented at the 22nd Shock and Vibration Symposium, Wright-Patterson AFB, Ohio, March 22-23, 1955; see _Supplement to Shock and Vibration Bulletin_ (U. S. Naval Research Laboratory), No. 22, pp. 24-27, July 1955.

5. Wheelwright, C. D., NASA Manned Spacecraft Center, Houston, Texas, Private Communication, July 1969 and January 1970.

6. Loriga, G., "Pneumatic Tools, Occupation and Health," _Encyclopedia of Hygiene, Pathology, and Social Welfare_, Vol. 2, International Labor Office, Geneva, 1934.

7. Ashe, W. F., Cook, W. T., and Old, J. W., "Raynaud's Phenomenon of Occupational Origin," _Archives of Environ. Health_, Vol. 5, October 1962, pp. 333-343.

8. Lance, G. N., "Motion of a Viscous Fluid in a Tube Which is Subjected to a Series of Pulses," _Quart. of Applied Math_, Vol. XIV, No. 3, 1956, pp. 312-315.

9. Roberts, L. B., Dines, J. H., "Physiological and Pathological Effects of Mechanical Vibration on Animals and Men," The Ohio State University, Department of Preventive Medicine, December 1965.

10. Pieper, H., "Catheter-tip Manometer for Measuring Blood Pressure During g-Changes," _J. Appl. Physiol._, Vol. 22, No. 2, February 1967, p. 352.

11. Clark, J. G., Williams, J. D., Hood, W. B., Murray, R. H., "Initial Cardiovascular Response to Low Frequency Whole Body Vibration in Humans and Animals," _Aerospace Medicine_, Vol. 38, May 1967, pp. 464-467.

12. Jones, R. T., "Applications of Fluid Dynamics to Circulatory Assist Devices," presented at the AIAA 3rd Annual Meeting, Boston, November 29-December 2, 1966. Paper No. 66-931.

13. Howe, J. T., NASA OART, Washington, D. C., Private Communication, October 1968.

14. Hooks, L. E., "An Analytical Model for Estimating Total Heart Power Output With and Without Whole-Body Vibration," Ph. D. Dissertation, The Ohio State University, 1970.

15. Hay, G. D., "The Effect of Longitudinal Whole-Body Vibration on Wave Propagation in the Aorta," Ph. D. Dissertation, The Ohio State University, 1972.

16. Womersley, J. R., "An Elastic Tube Theory of Pulse Transmission and Oscillatory Flow in Mammalian Arteries," Wright Air Development Center, WADC Report TR 56-614, 1957.

17. Schlichting, H., Boundary Layer Theory, McGraw Hill, New York, 1968.

18. Rudinger, G., "Review of Current Mathematical Methods for the Analysis of Blood Flow," ASME Symposium on Biomedical Fluid Mechanics, 1966, p. 1.

19. Hooks, L. E., Nerem, R. M., and Benson, T. J., "A Momentum Integral Solution for Pulsatile Flow in a Rigid Tube With and Without Longitudinal Vibration," presented at the Engineering Science in Biomedicine Meeting of the Society of Engineering Science, Washington University, St. Louis, November 3-5 1969; also Int. J. Engr. Sci., Vol. 10, 1972, pp. 989-1007.

20. Nerem, R. M., "Vibration-Induced Arterial Shear Stress," Arch. Environ. Health, Vol. 26, 1973, pp. 105-110.

21. Fry, D. L., "Acute Endothelial Changes Associated With Increased Blood Velocity Gradients," Circulation Research, Vol. 22, 1968, p. 165.

22. Baker, T. W., Allen, E. V., "Diagnostic Value of Arteriography," Report of Case Proc. Staff Meeting, Mayo Clinic, Vol. II, 1936, p. 500.

23. Barker, N. W., and Hines, E. A. "Arterial Occlusion in Hands and Fingers Associated With Repeated Occupational Trauma," Proc. Staff Meeting, Mayo Clinic, Vol. 19, 1944, pp. 345-349.

APPENDIX - PUBLICATIONS AND PRESENTATIONS

1. Nerem, R. M., "Fluid Mechanical Aspects of Blood Flow," _Proceedings of the 9th International Symposium on Space Technology and Science_, pp. 1201-1216 (Tokyo, Japan, August 25-30, 1969).

2. Hooks L. E., Nerem, R. M., and Benson, T. J., "A Momentum Integral Solution for Pulsatile Flow in a Rigid Tube With and Without Longitudinal Vibration," presented at the Engineering Science in Biomedical Meeting of the Society of Engineering Science, Washington University, St. Louis, November 3-5, 1969.

3. Nerem, R. M., "Some Aspects of Cardiovascular Behavior During Low-Frequency Vibration," _Proceedings of the AGARD Specialists, Meeting on Fluid Dynamics of Blood Circulation and Respiratory Flow_, held at Naples, Italy, May 4-6, 1970.

4. Nerem, R. M., "A Look at Cardiovascular Behavior During Whole-Body Vibration from a Fluid Mechanical Viewpoint," AIAA Fluid and Plasma Dynamics Conference, Los Angeles, California, June 29-July 1, 1970 (presented by J. T. Howe).

5. Hooks, L. E., "An Analytical Model for Estimating Total Heart Power Output With and Without Whole-Body Vibration," Ph. D. Dissertation, The Ohio State University, 1970.

6. Nerem, R. M., "Vibration-Induced Arterial Shear Stresses," _The Lancet_, January 9, 1971, p. 78.

7. Hooks, L. E., "An Analytical Model for Estimating Total Heart Power Expenditure With and Without Whole-Body Vibration," _Proceedings of the Third Annual Houston Conference on Computer and System Science_, held at University of Houston, Texas, April 26-27, 1971.

8. Nerem, R. M., "Vibration-Induced Arterial Shear Stress and Raynaud's Phenomenon of Occupational Origin," 24th Annual Meeting, American Academy of Occupational Medicine, Pittsburgh, Pa., February 9-11, 1972.

9. Nerem, R. M., "Arterial Wall Shear Stress Induced by Vibrating Hand Tools," _Proceedings of the 25th Annual Conference on Engineering in Medicine and Biology_, held at Bal Harbor, Fla., October 1-5, 1972, p. 8.

10. Hay, G. D., Nerem, R. M., and Hamlin, R. L., "Low Frequency Whole-Body Vibration Effects on Pulse Wave Propagation," _Proceedings of the 25th Annual Conference on Engineering in Medicine and Biology_, held at Bal Harbor, Fla., October 1-5, 1972, p. 217.

11. Hooks, L. E., Nerem, R. M., and Benson, T. J., "Momentum Integral Solution for Pulsatile Flow in a Rigid Tube With and Without Vibration," Int. J. Eng. Sci., Vol. 10, 1972, pp. 989-1007.

12. Hay, G. D., "The Effect of Longitudinal Whole-Body Vibration on Propagation in the Aorta," Ph. D. Dissertation , The Ohio State University, 1972.

13. Nerem, R. M., "Vibration-Induced Arterial Shear Stress," Arch. Environ. Health, Vol. 26, 1973, pp. 105-110.