The Role of the Peripheral Vascular Bed in Arterial Pulse Wave Propagation

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ABSTRACT

An analytical model was developed to simulate human arterial pulse wave propagation in major arteries. It was verified with an experimental model. The models investigated the effects of the peripheral vascular bed on pressure pulse morphology. The models revealed that the capillary bed in normal humans acts as a high resistance, low reflectance, boundary condition.

INTRODUCTION

The human arterial system represents a complex fluid mechanics problem. The flow is unsteady and periodic. It is generated by the heart, a complex, time varying, flow source. This pumping device feeds the arteries, a system of branching and tapering tubes. The arteries empty into the venous system. This exchange takes place through a fine grid of very small diameter tubes, the capillaries.

Pressure and flow waves have been monitored with catheter transducers and electromagnetic flowmeters in several different arteries [1]. There is an alteration in shape of the waveform with increasing distance from the heart. For example, the pressure amplitude increases with distance from the heart but the mean pressure decreases. In addition, the diastolic wave becomes more identifiable with increasing distance from the heart.

In contrast to the pressure waves, the amplitude of the flow waves decrease, and the period of reverse flow disappears as the distance from the heart increases.

Many investigators have attributed the variation in pressure wave shape with distance to wave reflection. It is believed that the peripheral vascular bed acts as a site of significant pulse wave reflection [1,2]. This wave reflection is widely assumed to be the cause of the diastolic wave.

This hypothesis was tested using analytical and experimental models of arterial pulse wave propagation developed specifically for this purpose. Different types of peripheral resistance devices were utilized and the amount of reflection/resistance was measured. Comparisons were made between pressure waves from the analytical and experimental models with those obtained from normal humans. These comparisons provided information about the role of the peripheral resistance in terms of wave propagation.

Background

Wave reflections in tubes occur at impedance discontinuities. These discontinuities may be the result of changes in tube material properties, fluid properties or cross-sectional area. Admittance is the property which governs the amount of wave energy which is reflected compared to that which is transmitted. It is can be shown to be the ratio of cross-sectional area to the product of wavespeed and fluid density [3].

$$Y = \frac{A}{a(\rho)} \qquad (1)$$

The type of reflection (positive or negative) which occurs at a junction of two dissimilar tubes may be calculated based on the admittances of the two tubes.

$$\text{Reflection} = \frac{Y_1 - Y_2}{Y_1 + Y_2} \qquad (2)$$

If the result of the calculation is a number with a positive value the wave is positively reflected. Alternatively, a negative value indicates a negative reflection. The amount of energy reflected is determined by the square of Eqn. 2.

$$\%\ \text{Energy Reflected} = \frac{(Y_1 - Y_2)^2}{(Y_1 + Y_2)^2} \qquad (3)$$

The information obtained from the use of these equations provided a basis of comparison for various peripheral vascular resistance models.

GOVERNING EQUATIONS

The analytical model simulated flow in a single artery. It was established from the conservation of mass and momentum equations for one-dimensional flow of a Newtonian fluid in a cylindrical tube. The flow was considered unsteady and periodic. The continuity and the momentum equations used were [4]

$$(\rho AV)_x + (\rho A)_t = 0 \qquad (4)$$

$$gH_x + VV_x + V_t \frac{fV|V|}{2D} = 0 \qquad (5)$$

In the continuity equation, a was the wavespeed which was calculated from

$$a^2 = \frac{\frac{K}{\rho}}{1 + \left[\left(\frac{2k}{E_R}\right)(1+\mu)\right]} \qquad (6)$$

The underlying assumption for this form of the wave speed was that cicumferential deformation is the dominant element in the compliance.

Viscous losses were approximated by the use of the Darcy-Weisbach equation [4]. This assumption is commonly employed in the study of hydraulic transients, since a better model of the viscous losses does not exist.

This model included the convective acceleration term in the momentum equation, an improvement over other analytical models. This term was omitted in previous studies to simplify the analysis [5].

The equations were solved using the method of characteristics. This method was chosen since it easily incorporates unsteady flow and nonlinearities. It has the advantage of being easily programmable and is capable of implementing many types of boundary conditions. The partial differential equations (Eqn. 4 and 5) were transformed by the method of characteristics into particular total differential equations. These equations were integrated to yield finite difference equations which were conveniently solved by a numerical technique.

Figure 1 shows a schematic of the system modeled in this study. The pressure head and velocity were determined at discrete intervals of time and space in a single compliant tube (the artery). The upstream boundary condition was a flow waveform. The downstream boundary condition was a peripheral resistance element. The input parameters for the analytical model were obtained from the experimental model.

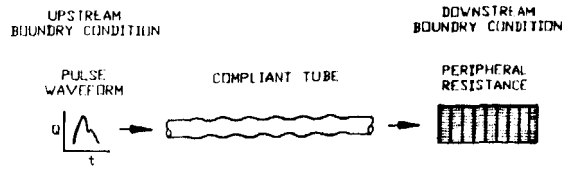

Figure 1: Schematic of Analytical Model

EXPERIMENTAL MODEL

An experimental model provided a means of verifying the analytical model. The model provided a desired input flow waveform through the use of a digitally controlled servo system.

Three types of peripheral resistance elements were tested to determine the role of the peripheral vascular bed. Each type of resistance element represented a different downstream impedance. The first type of resistance element used was a sudden expansion into a head tank. This represented a system with a constant downstream pressure, but allowing for reflection. The second type of element used was a manifold of capillary tubes. These were intended to simulate the geometry of the peripheral vascular bed. The element provided a large resistance and was also a significant wave reflector. Finally, the third type of resistance element utilized was a very long tube (1500 cm). This element provided a high resistance without allowing for significant reflection.

RESULTS AND DISCUSSION

Comparisons were made between the pressure waves obtained from the experimental model and a physiologic waveform from the literature [1]. These comparisons are shown in Figure 2 for each of the three downstream impedance elements. Figure 2c shows that the experimental pressure waveforms obtained with the long tube impedance element provided the closest simulation to a normal human waveform. The other elements may have been of the correct amplitude but had an incorrect morphology. It is clear that, for a fixed input flow, the downstream boundary condition establishes the wave morphology.

The parameters from the experiment which used a long tube downstream impedance were used as input to the analytical model. A comparison of analytical, experimental and clinical pressure waveforms is shown in Figure 3. It can be seen that the waveforms had the same slopes and total amplitudes. The timing of the primary and secondary waves was exactly the same for the three models. The amplitude of the secondary wave was somewhat greater for the analytical model as compared to the experimental and clinical data. The analytic model predicted a secondary pulse amplitude which was 15 mm Hg greater than the experimental and clinical waves. This is most likely the result of the constant value used for the friction factor. It is known that in unsteady flow, however, the friction factor is of varying magnitude [6,7].

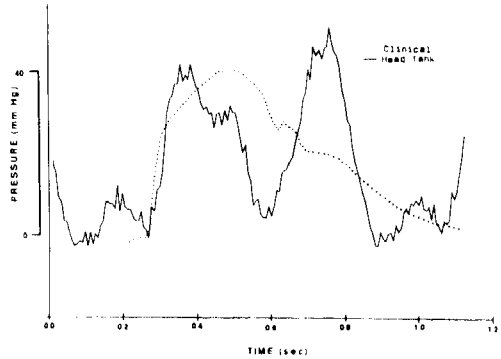

Figure 2a: Experiment vs Clinical - Head Tank

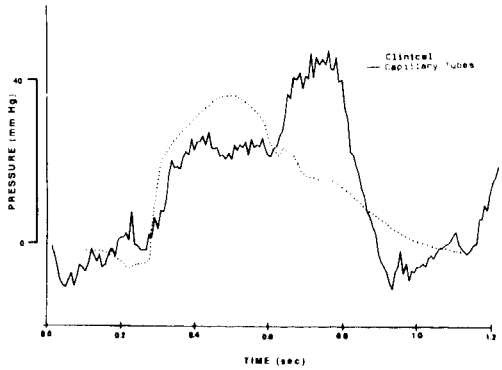

Figure 2b: Experiment vs Clinical - Capillary Tubes

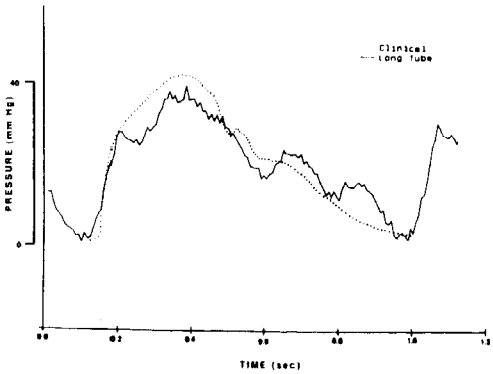

Figure 2c: Experiment vs Clinical - Long Tube

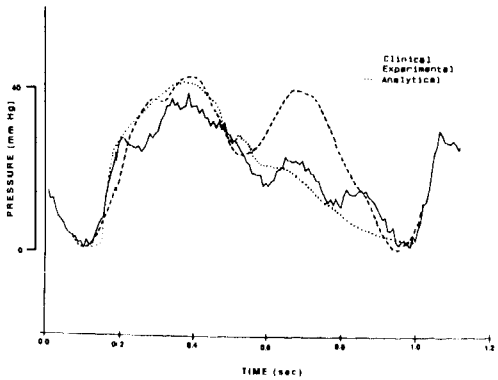

Figure 3: Experiment vs Clinical vs Analytical

The amount of energy reflected was calculated for the downstream impedance elements used in the experimental model (using Eqn. 3). These values were compared to estimates from junctions in the arterial system [8,9]. The values are summarized in Table 1.

TABLE 1: Energy Reflected at Peripheral Resistance Elements

Experimental/Analytical

Resistance Element	Pos./Neg. Reflection	% Reflected
Head Tank	-	99
Capillary Tubes	+	73
Long Tube	+	28

Clinical

Junction	Pos./Neg. Reflection	% Reflected
Iliac Bifurcation	+	12
Large artery/Arteriole	-	0.01
Largest artery/Arteriole	+	28

The table of clinical data shows that the arterial system has almost no reflection at any junctions. The "worst case" scenario of the aorta connected dirctly to an arteriole only showed positive reflection of 28%. Fortuitously, this is the same value obtained for the experimental system with a long tube impedance element. Reflection is discernable only when it is above about 40% [3]. A value of only 28% means that essentially no reflected waves can be identified in the observed waveforms.

The fact that the arterial system possesses branches and is tapered can only reduce wave reflection. The tapering allows diameter and compliance to change gradually. This results in only slight variations in admittance and hence, no site for wave reflection. The most significant bifurcation, the iliac bifurcation, is only a 12% positive reflection site. This represents the greatest impedance mismatch in the arterial system due to branching. The thoracic artery branches are almost perfectly matched [3]. The lower extremity branches were calculated to have significantly less reflection than the iliac bifurcation.

These results cast serious doubt on the presumption that normal arterial pulse wave morphology is caused by pulse reflection. The only source of significant wave reflection is an arterial obstruction.

The normal artery simulations thus revealed that the arterial system can be adequately modeled with a system that has uniform compliance, little or no peripheral reflection, no branching and no tapering. This represents a much simpler model than previously thought necessary. It was previously assumed that a satisfactory analytical model required branching, nonlinear compliance and vessel tapering. This research has shown that these complexities are not

necessary. A close look at the basic physics of the fluid system, rather that the intricate geometry of the arterial system, has led to a much more tractable but realistic problem.

CONCLUSIONS

The method of characteristics forms a suitable basis for an analytical model which can be used to simulate arterial flow. The model can include nonlinear effects in both the convective and viscous terms. It was shown that a model of the arterial system should have little or no downstream reflection and does not require any branching or vessel tapering.

The study has shown that the peripheral vascular bed in a normal human acts as a highly resistant wave absorber and is not a significant source of wave reflection.

The analytical model developed in this study can be used to study arterial disease or other normal pathological states such as vasoconstriction or vasodilitation. Parameters can be varied much more easily and economically in an analytical model than in an experimental model. The versatility of this analytical model provides a valuable tool for understanding the complexities of the arterial system.

NOMENCLATURE

Y = admittance
a = wave speed
K = fluid bulk modulus
A = area
H = pressure head
V = velocity
f = friction factor
D = diameter
μ = poisson's ratio
E_r = Modulus of rigidity

Subscripts
x = derivative with respect to distance

t = derivative with respect to time

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