Pulse Wave Propagation

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SUMMARY This report evaluates pulse wave propagation with respect to contributions by vascular wall elastic and geometric properties, vessel wall and blood viscosity, and nonlinearities in system parameters and in the equations of motion. Discrepancies in results obtained with different experimental methods and theory are discussed and resolved. A three-point pressure technique was used to obtain measurements from the abdominal aorta, carotid, iliac, and femoral arteries of dogs. Computations involved linear, as well as nonlinear methods. Results are presented along a continuous path of transmission (abdominal aorta, iliac, femoral), and it is shown that variations in phase velocity can be explained entirely by the geometric variation of these vessels. Phase velocities are shown to be frequency independent at 54 Hz whereas attenuation increases progressively for higher frequencies. Determination of propagation coefficients using maximal, compounded values of reported viscoelastic and geometric properties just manages to span the range of phase velocities, determined in different laboratories, but does not do so for attenuation. Also, differences in experimental techniques cannot explain these discrepancies. Consideration of geometric taper, nonlinear compliance, all the terms in the equation of motion, and the effect of wall and blood viscosity resolves discrepancies between theoretical models and experimentally derived phenomena. Circ Res 49: 442-452, 1981

PULSES originating at the left ventricle are modified as they propagate towards the periphery. This is attributed to the effects of blood viscosity, to arterial viscoelasticity resulting in frequency dispersion and selective attenuation, and to site-dependent summation of incident and reflected pulses.

Euler (1775) provided the initial theoretical study of pulse propagation. Until 1914, his work and that of others emphasized the inertial property of blood and arterial wall compliance. In this period the Moens-Korteweg expression for pulse wave velocity [a relationship originally derived by Young (1809)] was established.

Viscous, as well as inertial, properties of blood were first included in an elastic tube model by Witzig (1914). Morgan and Kiely (1954), then Womersley (1957), extended Witzig’s work by replacing the purely elastic wall with a simple viscoelastic wall. Lacking experimental data, Jager et al. (1965) introduced a thick, but wholly elastic wall. Subsequently, Cox (1968) replaced Jager’s axially constrained wall by a wall free of such constraint and included viscoelastic properties in a more general form than had Morgan, Kiely, or Womersley. Viscoelastic wall properties, encompassing stress-relaxation, creep, and hysteresis, were treated extensively by Westerhof and Noordergraaf (1970).

Concurrently, concepts advanced from a simple elastic tube representing the arterial system to branching patterns generating smaller vessels with increasing total cross-sectional area. Taylor (1966a, 1966b) investigated a randomly distributed, dichotomously branching system of elastic vessels. Westerhof and Noordergraaf (1970), as well as others, evaluated the role of viscoelastic properties in the human arterial system.

The validity of the Westerhof model was established by comparison with measured pressure and flow waveforms and by derived variables such as input impedance and frequency-dependent wave velocity. Concurrence with measured variables in the systemic arteries lent credence to a linear transmission concept more general than its predecessors, e.g., the windkessel and single tube, while including these as special cases. Theory accounted for modification, with propagation, of pressure and flow pulses (Noordergraaf, 1978).

Milnor and Nichols (1975) indicated discrepancies in the propagation constant (γ) between experiment-based calculations and the Westerhof theory. Later, however, results obtained by Wetterer et al. (1978) supported the theory. A second evaluation by Milnor and Bertram (1978), employing similar techniques as did Milnor and Nichols (1975), confirmed the earlier experimental work. On this basis they suggested the inadequacy of current theoretical concepts. Subsequently, Busse et al. (1979), utilizing the same techniques as Wetterer et al. (1978), reconfirmed the Wetterer report, again supporting the theory. Busse et al. (1979) comment on the experimental technique used by Milnor and others as being less than optimal. The need for independent assessment of γ is clear.

In this report we investigate γ, a parameter embodying basic circulatory system characteristics. γ is separable into two components, i.e.,

\[ \gamma = \alpha + j\beta \]  

(1)
where
\[ \beta = \omega/c. \] (2)

\( \alpha \) denotes the attenuation coefficient, \( \beta \) the phase coefficient, and \( j = \sqrt{-1} \). \( c \) is the phase velocity, i.e., the speed of propagation of a frequency component of the pulse, free of reflection, \( \omega = 2\pi f \), and \( f \) is the frequency. Theory encompasses a propagation constant that is frequency dependent, as well as arterial properties dependent. Several techniques permit evaluation of \( \gamma \) via in vivo measurements. These are summarized in a previous paper (Li et al., 1980a).

A three-point pressure technique (Li et al., 1980a) was utilized for the results reported here. Published data are compared, the bases for results disclosed, sources of discrepancies identified, and the differences resolved between experimental groups and between experiments and theory.

**Methods**

Six mongrel dogs of either sex weighing 18–22 kg provided data. Anesthesia was induced with thiopental sodium (0.5 ml/kg) and, after tracheal intubation, maintained with an oxygen-halothane (0.5–2.5%) mixture. An iv saline drip was established and the lead II ECG monitored.

Experiments were performed on the carotid and femoral arteries, on the abdominal aorta, and on the iliac artery of each dog. Branches along the vessel segment under investigation were ligated as was the short continuation segment of the abdominal aorta to the internal iliac arteries. Vessels were kept moist with physiologic saline.

Side-port pressure was measured with a manometer system described previously (Li et al., 1978). The system consisted of Statham P23Gb transducers, each connected to a 5-cm modified 22G needle.

Frequency responses were flat to over 30 Hz, with negligible phase shift (<0.1°). The systems were filled with preboiled water or heparinized saline solution. The manometer system was oriented in space by a stereotaxic device. Each needle was placed in the vessel lumen close to the wall. Needle separation (2–3 cm) was measured with a precision gauge achieving a resolution of better than 0.25 mm. When all pressure channels demonstrated similar characteristics and steady state, at least 3 minutes of data were recorded with the ECG signal on an FM recorder.

Recorded data were replayed (error < 1%) and viewed on a storage oscilloscope. Sections of data with minimal beat-to-beat variation over a respiratory cycle were amplified and input to a digital computer. Signal/noise ratio was improved and round-off errors reduced by eliminating DC signals and amplifying and sampling the oscillatory portion of the pressure pulses. Signals were sampled at 500 Hz. Aliasing was eliminated in the digitized data by low pass filtering at a cut-off frequency of either 25 or 50 Hz (fifth-order Lerner filter), depending on the noise level. Much of the high frequency noise contamination (especially 60 Hz) was thus eliminated. Since final computation of attenuation and phase coefficients utilized only ratios of signals, low frequency tape flutter, which influences all channels equally, was minimized.

Results depend on the resolution of small pressure differences, and are thus sensitive to imbalance among the transducers. Li et al. (1978) reported a technique utilizing mean pressure as a reference to eliminate most imbalance problems inherent with pressure transducers. The technique achieves an accuracy comparable to, if not better than, those derived with more elaborate methods.

Blood pressure varied among dogs between 88–116 mm Hg (mean = 104.2) and heart rate varied between 83–100 beats/min (mean = 90.5). From pooled data, harmonics of comparable frequencies were averaged over a bandwidth of 1.6 Hz (=2Δf). Thus, for the nth harmonic, the frequency band is \( f_n = f_m \pm \Delta f \), with a center frequency of \( f_m = n \times 1.6 \) Hz. With one exception, heart rate variation among dogs was small so that a narrow bandwidth was feasible. Fourier components of pressure signals were computed to the 12th harmonic.

The real and imaginary parts of the propagation constant were computed utilizing
\[ \gamma = \frac{1}{\Delta z} \cosh^{-1} \left( \frac{p_1 + p_2}{2p_2} \right) \] (3)

where \( \Delta z \) is the distance between the pressure measurement sites for \( p_1, p_2 \), and \( p_3 \).

**Results**

Phase velocities (Fig. 1) for the abdominal aorta, iliac artery, femoral artery, and carotid artery were each averaged for all dogs. These phase velocities are frequency dependent and patterned similarly regardless of vessel size. Mean values for the phase velocity asymptotes were 6.66 m/sec for the abdominal aorta, 7.65 m/sec for the iliac artery, 8.04 m/sec for the carotid artery, and 8.78 m/sec for the femoral artery. Thus phase velocity increases as the pulse wave travels from the central to the peripheral arteries. The mildly dispersive nature of the pulse components is apparent from the frequency dependence of phase velocity (Eq. 2).

Figure 2 summarizes results for attenuation coefficients. Attenuation increases with frequency and with diminishing vascular size. For low frequencies, e.g., about 1.5 Hz, the femoral artery manifests highest attenuation, i.e., 1.6 m\(^{-1}\); the carotid artery, 1.1 m\(^{-1}\); the iliac artery, 1.0 m\(^{-1}\); and the abdominal aorta lowest attenuation, i.e., 0.7 m\(^{-1}\). At high frequencies (>8 Hz) damping was found to be similar in the femoral, carotid, and iliac arteries. The abdominal aorta, however, maintained a lower attenuation coefficient.
The attenuation coefficient, reported here for the first time along a continuous path of wave transmission (i.e., the lower abdominal aorta, the iliac artery, and the femoral artery), reflects the effect of damping along the path. At 7 cm above the aorto-iliac junction a fundamental amplitude of 1.00, of a low frequency pulse (1.6 Hz), is attenuated to 0.95 upon reaching the junction and to 0.89 upon leaving the external iliac artery. For a 9.6-Hz component, the corresponding values, normalized with respect to the initial fundamental amplitude, are 0.77, 0.53, and 0.35, demonstrating greater attenuation of high frequency components. Thus, this path resembles a low pass filter with a long time constant. Such damping of the antegrade pulse applies also to the retrograde pulse.

**Discussion**

**γ According to Linear Theory**

Questions concerning the adequacy of linear theory and experimental techniques for the development of γ (Eqs. 1 and 2) require an appraisal of its contributors. All equations, on which both models and experimental data evaluation are founded, are linearized and all are equivalent expressions of the linearized Navier-Stokes equations and equation of continuity. With linearization, the equation which describes the motion of blood assumes the nonlinear terms to be insignificant. Also, wall properties, specifically stress-strain relationships, are treated as constant within the range of operating pressure.

From the Westerhof model (1970), the phase velocity and attenuation coefficient may be derived in the following form

\[ c = \frac{\omega}{\beta} = \frac{c_0 (q_1^2 + q_2^2)^{1/4} (1 + \tan^2 \psi)^{1/4}}{\cos(\phi/2)} \]  

(4)

\[ \alpha = \frac{\omega \sin(\phi/2)}{c_0 (q_1^2 + q_2^2)^{1/4} (1 + \tan^2 \psi)^{1/4}} \]  

(5)

where

\[ c_0^2 = \frac{Eh (2r_1 + h)}{3 \rho (r_1 + h)^3}. \]  

(6)

\[ \alpha' = (\omega \rho / \mu)^{1/2} r_1. \]  

(7)

Here \( \mu \) is blood viscosity and \( J_0 \) and \( J_1 \) are Bessel functions of the first kind of zero and first order, respectively. \( q_1 \) and \( q_2 \) reflect the inertial and resistive properties, respectively, of the fluid (Womersley, 1957, Table 4).
Psi is the angle defined by real and imaginary parts of the complex number defining viscoelastic wall properties and

\[ \phi = \tan^{-1} \frac{q_2/q_1 + \tan \psi}{1 - q_2/q_1 \tan \psi} \]  

(8)

Comparison of Phase Velocity (c) in Reported Data

The frequency-dependent pattern of wave velocity (as displayed in Figure 1) was predicted implicitly by Witzig as early as 1914. Subsequent theories express the relationship explicitly (Noordergraaf, 1969).

Figure 1 provides, apparently, the sole reported data of the frequency spectrum of phase velocity for the lower abdominal aorta and iliac artery. For the femoral artery, the frequency spectrum of the phase velocity was provided by Gessner and Bergel (1966), Cox (1971), Milnor and Nichols (1975), and by the present authors. Similar data are also presented for the carotid artery. The various authors report comparable wave velocities derived by different means and these data are listed in Table 1. Often, only single values have been reported.

Regional Alteration of Phase Velocity

Phase velocity is observed to increase peripherally and to follow a consistent pattern with respect to its dependence on frequency. This increase in velocity may be caused by variation of vascular properties.

Representation of viscoelastic and geometric properties in the phase velocity is seen in Equation 4 in \( c_0 \), \( q_1 \), \( q_2 \), and \( \psi, q_1 \), and \( q_2 \) are geometry dependent through Equation 7. The geometric influence on the factors \( q_1 \) and \( q_2 \), however, can be shown to be small for the carotid, abdominal aorta, iliac, and femoral arteries. This influence reduces phase velocity slightly in the more peripheral vessels for a wide range of \( \psi \) and \( \omega \). Since phase velocity increases peripherally, the dominant factors must reside in \( c_0 \).
**Table 1** Comparison of Reported Wave Velocity

<table>
<thead>
<tr>
<th>Vessel</th>
<th>Velocity (m/sec)</th>
<th>Data source</th>
<th>Method</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdominal aorta</td>
<td>6–8</td>
<td>Dog</td>
<td>f-f</td>
<td>Dow and Hamilton (1939)</td>
</tr>
<tr>
<td></td>
<td>5.5–8.5</td>
<td>Dog</td>
<td>f-f</td>
<td>Lasz and Müller (1952)</td>
</tr>
<tr>
<td></td>
<td>9.3</td>
<td>Dog</td>
<td>dE, mv‡</td>
<td>Bergel (1961b)</td>
</tr>
<tr>
<td></td>
<td>6.7–7.4</td>
<td>Dog</td>
<td>f-f</td>
<td>McDonald (1968)</td>
</tr>
<tr>
<td></td>
<td>6.7</td>
<td>Dog</td>
<td>mv</td>
<td>Present authors</td>
</tr>
<tr>
<td>Iliac artery</td>
<td>7</td>
<td>Young men</td>
<td>dE</td>
<td>Learoyd and Taylor (1966)</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>Old men</td>
<td>dE</td>
<td>Learoyd and Taylor (1966)</td>
</tr>
<tr>
<td></td>
<td>7–8</td>
<td>Dog</td>
<td>f-f</td>
<td>McDonald (1968)</td>
</tr>
<tr>
<td></td>
<td>7.7</td>
<td>Dog</td>
<td>mv</td>
<td>Present authors</td>
</tr>
<tr>
<td>Femoral artery</td>
<td>8–12</td>
<td>Dog</td>
<td>f-f</td>
<td>Dow and Hamilton (1939)</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>Man</td>
<td>f-f</td>
<td>Kapal et al. (1951)</td>
</tr>
<tr>
<td></td>
<td>8.5–13</td>
<td>Dog</td>
<td>f-f</td>
<td>Lasz and Müller (1962)</td>
</tr>
<tr>
<td></td>
<td>9.3</td>
<td>Dog</td>
<td>dE, mv</td>
<td>Bergel (1961b)</td>
</tr>
<tr>
<td></td>
<td>18</td>
<td>Young men</td>
<td>f-f</td>
<td>Learoyd and Taylor (1966)</td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>Old men</td>
<td>f-f</td>
<td>Learoyd and Taylor (1966)</td>
</tr>
<tr>
<td></td>
<td>8± 1.1</td>
<td>Dog</td>
<td>mv</td>
<td>Gessner and Bergel (1966)</td>
</tr>
<tr>
<td></td>
<td>8.3–10.3</td>
<td>Dog</td>
<td>f-f</td>
<td>McDonald (1968)</td>
</tr>
<tr>
<td></td>
<td>8.2</td>
<td>Dog</td>
<td>mv</td>
<td>Cox (1971)</td>
</tr>
<tr>
<td></td>
<td>10.4± 0.4</td>
<td>Dog</td>
<td>dE</td>
<td>Cox (1975)</td>
</tr>
<tr>
<td></td>
<td>8.5</td>
<td>Dog</td>
<td>mv</td>
<td>Milnor and Nichols (1975)</td>
</tr>
<tr>
<td></td>
<td>8.8</td>
<td>Dog</td>
<td>mv</td>
<td>Present authors</td>
</tr>
<tr>
<td>Carotid artery</td>
<td>5–12</td>
<td>Man</td>
<td>f-f</td>
<td>Bramwell et al. (1922)</td>
</tr>
<tr>
<td></td>
<td>9.4</td>
<td>Dog</td>
<td>dE, mv</td>
<td>Bergel (1961b)</td>
</tr>
<tr>
<td></td>
<td>6.1–7.4</td>
<td>(Proximal) dog</td>
<td>f-f</td>
<td>McDonald (1968)</td>
</tr>
<tr>
<td></td>
<td>7.7–8</td>
<td>(Distal) dog</td>
<td>f-f</td>
<td>McDonald (1968)</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>Dog</td>
<td>mv</td>
<td>Present authors</td>
</tr>
</tbody>
</table>

* f-f, measured as foot-foot velocity.
‡ dE, calculated utilizing dynamic elastic modulus.
MV, mean value from frequency spectrum (≥4 Hz) of phase velocity.

McDonald (1974) reports wall properties for these vessels in terms of elastin:collagen ratios; the ratios are remarkably consistent. On this basis, if one assumes that E is essentially constant among these vessels, the effect of geometric variation can be examined. Measurements by the present authors, in conjunction with this assumption, yielded the following values of c (normalized to the respective value obtained for the abdominal aorta): iliac: abdominal: experimental, 1.15, computed, 1.24; femoral: abdominal: experimental, 1.32, computed, 1.24; carotid: abdominal experimental, 1.21, computed, 1.20. Thus variation in phase velocity among these vessels can be explained by their geometric variation.

**Variation of Phase Velocity with System Parameters**

Discrepancies in phase velocity reported by various authors may be due to a number of factors. One of these factors may relate to variation in values of parameters utilized in the determination.

If measured pressures and flows are utilized in the transmission expressions, c and α are derived directly without knowledge of wall properties, e.g., Equations 1–3. Determination, however, of these pulse propagation variables, utilizing, e.g., the Westerhof model (Eqs. 4 and 5), requires knowledge of wall properties. These are derivable from pressure-diameter relationships whose real part is E and whose imaginary part (frequency-dependent viscous component) in conjunction with E determines ψ.

E is related to the level of transmural pressure in a nonlinear manner, to its time rate of change, and to the level of smooth muscle activation (Bergel, 1961a, 1961b; Gow, 1968, 1970; Cox, 1975, 1978; Bauer et al., 1979; Monos et al., 1979). Consequently, heart rate, mean pressure, animal state, accuracy of measurement, and the sensitivity of phase velocity and attenuation to variation in E become considerations.

In the femoral artery, for example, in the region of 140 mm Hg, a pulse pressure of approximately 10 mm Hg can result in variation in E of the order of 25%. Variations in calculated phase velocity (utilizing linear theory which makes no allowance for dependence of E on pressure) of the order of 2% can occur with a 1% difference in transmural pressure. Greater variation occurs at higher pressures.

Pulse pressure in the femoral artery may be in the order of 50 mm Hg. Since higher pressures exist for a relatively short period, c₀ may be expected to be lower for a greater portion of the cycle because of the pressure dependence of E. Thus, when determination of phase velocity is based on measurements (not utilizing E), one would expect it to tend towards its lower extreme. Phase velocity, computed from experimental data without introducing E explicitly (Eq. 3), results as a consequence of all
variations in E occurring during the measurement period. In contrast, c as calculated via the model (Eq. 4) reflects the particular E chosen. Any comparison of measured and calculated (model) values should, ideally, be made with an E equivalent to its composite value during the cycle.

Vessel dimensions also affect c (Eq. 6). A range of h/r, between 0.11-0.29 for the femoral artery has been reported in the literature (Bergel, 1961a; Peterson, 1964; Cox, 1971, 1978). This range of values can alone produce variations in calculated phase velocity of up to about 20%. These dimensions are also related to the level of transmural pressure. In the femoral artery, variation in E could be easily as high as 150% over a range of 50 mm Hg and the h/r ratio in the order of 10%. From consideration of both variations, calculated values of c can differ up to 97% at low frequencies and up to 80% at high frequencies.

Phase velocity computed via the model is additionally dependent on the phase angle (ϕ). Theoretically, this angle can vary from zero, for the purely elastic wall, to π/2 radians for the purely viscous wall. Thus, ϕ generates frequency-dependent scale factors affecting c (Eqs. 4 and 8) which, theoretically, can range between 1 and ∞; i.e., if a wall is entirely viscous, it acts as a stiff wall and c can theoretically reach ∞ (Li et al., 1980a; Melbin et al., in press). Since the wall is viscoelastic, this effect is constrained.

For both low and high frequencies (e.g., 1 and 10 Hz, respectively), for small ϕ, variation in ϕ results in small alteration of c. For large ϕ, however, small variations in ϕ result in considerably greater alteration in c. Since reported values of ϕ for arteries considered herein are low, c is, in actuality, only moderately dependent on wall properties.

Figure 3a illustrates data of Milnor and Nichols (1975), Milnor and Bertram (1978), and the present authors. The solid line represents the phase velocity calculated from the Westerhof model (Eq. 4) using the parametric data of Milnor and Bertram (1978). The shaded region illustrates the range possible in the femoral artery with maximal reported variation in ϕ, E, r, and h. Imposition of extreme values of the parameters (found in the literature) just manages to encompass the values determined by the different investigators.

Variation of Attenuation with System Parameters

The available reports concerned with attenuation are limited to canine carotid and femoral arteries. These data also are not in complete agreement.

Figure 4 summarizes attenuation for the carotid artery, expressed in terms of transmission over a 10-cm distance. Illustrated are: values calculated from Womersley’s theory (1957) which accounts for blood viscosity, but assumes the arterial wall to be purely elastic; values calculated from Bergel’s in vitro experiments (1961b) which include viscoelas-

![Figure 3](http://circres.ahajournals.org/)

**Figure 3** Comparison of phase velocity (a) and attenuation (b) in the femoral artery, with variation in calculations, experimental methods, and parameter values.

- ■: data from Milnor and Nichols (1975); ▲: data from Milnor and Bertram (1978); ○: data from present authors. a: range of c possible with maximal reported variation in ϕ, E, r, and h is defined by the shaded region. Solid line is calculated using the Westerhof model (Eq. 5). b: range of α possible with maximal reported variation in ϕ, E, r, and h is defined by the shaded region. ——: blood viscosity alone; ····: wall viscosity alone; ———: transmission with combined blood wall viscosity (Bergel, 1961b); ———: present authors, using linear theory and linearized physical model; ———: present authors, using nonlinear theory and nonlinear physical model parameters.

The results depicted in Figure 4 indicate that damping is greater when both viscous effects of blood and viscoelastic effects of the arterial wall contribute. Thus, a perfectly elastic tube underestimates damping.

The results from in vivo experiments reported by Wetterer and co-workers are distinct from in vivo results reported by McDonald and Gessner and the
present authors, in that their data compare more favorably with attenuation predicted by theoretical models. These investigators do not provide an explanation for the discrepancy (about a factor of 4 in α) between their results and measurements of others, but do consider their two-point method a superior technique (Busse et al., 1979; also see introductory paragraphs).

Figure 5 summarizes the available data for attenuation in the femoral artery. The two series obtained by Milnor et al. (1975, 1978) from in vivo measurements, utilizing two pressure and two flow sensors, and those obtained by the present authors, employing a three-point pressure technique, are in close agreement. Both groups observed significantly greater damping than that predicted theoretically by the Womersley and Westerhof models. Experimental data obtained via the two-point method (Wetterer et al., 1978; Busse et al., 1979) are not available for the femoral artery.

Milnor and Bertram (1978) compare the results of their measurements to model calculations, and these authors suggest that the discrepancies may be due to the omission, in the model, of nonlinear terms from the Navier-Stokes equation. Since the same linearizing assumptions underlie both the model and their experiment-based calculation, utilization of the latter to form conclusions concerning the former is inconsistent.

An examination of Equations 4–8 indicates that α is subject to variations of ψ, E, and hν1 in much the same manner as is c. Although discrepancies in reported values of c (Fig. 3a) fall within the range developed by varying the parameters, discrepancies in a α between model and experiment exceed the boundaries of this range (Fig. 3b).

An additional feature of α (Ref. Eq. 6) not found with c is that, with increasing ψ, α varies from a finite value through a maximum, reaching zero at α = π/2. Pulse attenuation, although first increasing with increase in wall viscosity, would eventually be reduced as wall viscosity dominates wall compliance (Li et al., 1980b; Melbin et al., in press).

**Experimental Techniques and Accuracy of Measurement**

Discrepancies between results, obtained by different methods, raised questions concerning techniques. Busse et al. (1979) point out that, with all techniques, one works close to the resolving power of the measuring system. Regardless of technique, however, resolution of γ is related to cosh(γΔz) (Eq. 3). The nature of this function is such that, as its argument (Δz) approaches small values, cosh(γΔz) becomes consistently less sensitive to change in γ. Busse et al. (1979) argue that, as one increases the distance (Δz) between measuring points, sensitivity is improved. Additionally, since accurate measurements are difficult to obtain, they suggest that preference should be given to the method requiring a minimum of measured variables.

On these bases, they consider their technique superior since only two pressures are required and there is the inherent advantage of greater distance available between measuring points. Although these issues are valid, the conclusion is not necessarily warranted for the following reason.

The two-pressure method utilizes a fully occluded vessel with one measurement obtained at the plane of occlusion. Such an occlusion carries with it an underlying assumption of doubling of pressure amplitude with zero phase shift at this
plane. Even if these assumptions apply, the occlusion experiment introduces a reflection coefficient ($\Gamma$) which not only acts to impose amplitude alterations but also to generate frequency-dependent, high apparent wave velocities. Thus differences in measured variables may be reduced in the same manner as if the probes were in closer proximity to each other. Methods utilizing a greater number of measurements do not introduce vessel occlusion.

To illustrate, consider two extremes where $\Gamma = 1$ and $\Delta z = 6 \text{ cm}$ (for the case of complete occlusion) and $\Gamma = 0$ with $\Delta z = 3 \text{ cm}$ (for no reflection) using realistic values for $\alpha$ and $\beta$ such as: at 1 Hz, $\alpha = \beta = 0.005$ and at 10 Hz, $\alpha = \beta = 0.05$. In the occlusive case, at 1 Hz, pressure amplitudes differ by about 0.05% and the phase difference is about 0.05°. At 10 Hz, the amplitude variation is in the order of 0.3% and the phase difference about 1.5°. For the non-occlusive case, at 1 Hz, pressures differ by about 0.6% and the phase difference is about 0.9°. At 10 Hz, the amplitude variation is about 7.8% and the phase difference is about 8.6°. Thus, although a greater demand on the resolution of the measuring device. In reality, reflection coefficients are known to be frequency, state, and location dependent. The present authors find that $\Gamma$, at about 1 Hz, may range from about 1 to 0.6 (vasoconstriction to vasodilation, respectively) and, at 10 Hz, from about 0.3 to 0.1, respectively.

With respect to measuring devices, Busse et al. (1979) point out the greater inaccuracy inherent in flow measurements as compared with pressure measurements. In spite of the variation in devices and techniques, we cannot conclude that this accounts for the differences in attenuation between one group (e.g., Westerhof and Noordergraaf, 1970; Busse et al., 1979; and Wetterer et al., 1978) and another (e.g., Milnor and Nichols, 1975; Milnor and Bertram, 1978; and the present report).

**Linear and Nonlinear Pulse Transmission**

Linear and nonlinear approaches are distinct in several respects. For example, in linear transmission expressions, the steady flow component is separable from oscillatory components in the Fourier series expansion and the system parameters are taken to be constants. Here the steady flow component does not play a role in the calculations involving pulse propagation. Flow, however, especially if the vessel is tapered and exhibits other varying parameters, may affect oscillatory components.

The two major forms of experiments (occlusive or no-flows vs. flow) reveal that, in the former case, the conditions underlying linear transmission expressions prevail. Consequently, it might be expected that measurement-based propagation coefficients, derived via the occlusive experiment, correlate well with those derived via linear models. This is indeed the case (Fig. 4). For the latter, however, viscous and inertial effects are likely to be more significant. Such effects tend to alter both viscous losses and local velocities (energy conversions) in the vessel which provides, for example, pressure data from which propagation constants are evaluated.

In the measurement of pressure with side-mounted probes, potential or pressure energy which has been converted to the kinetic form will not be represented. Thus, if measured pressures, as utilized for example in Equation 3, are reduced due to energy conversion to the kinetic form, it will appear as though conversion was to the thermal form; i.e., diminution at the downstream measurement sites will be attributed to attenuation (viscous losses). Conversely, if there are conversions from kinetic to potential energy, it will appear as less attenuation, i.e., apparent reduced thermal losses. Additionally, in a vessel of altering cross-section (spatially and temporally), attenuation is site dependent, a phenomenon not recognized by linear transmission representations.

Solution of the full (nonlinear) equations of motion (Navier-Stokes) and continuity (Melbin and Noordergraaf, in press) was utilized to evaluate these issues. This was accomplished via the following theoretical experiment.

Based on data drawn from the literature or from independent measurements, a model for the femoral artery was designed. An equivalent correlate was also established whose parameters were mean values of the femoral model. Both were considered circular in cross-section. These two models served as conduits through which, with the aid of a digital computer, a pulse was transmitted in the time domain, according to the full solution of the equations of motion and continuity for the former and the linearized equations of motion and continuity for the latter. The input pulses, as well as pulses emerging 10 cm downstream, were converted by Fast Fourier Transform and the moduli of the first eight harmonics compared with those determined by the various workers utilizing linear transmission expressions. Each term in the Navier-Stokes equation was noted for each vessel.

The parameters for the two vessels are listed in Table 2. Vessel taper was expressed in conventional form as $r_1 = r_0 e^{-kz}$, where $r_0$ is the inner radius, $r_1$ is at the upstream end, $z$ is the axial distance along the vessel, and $k$ is a taper constant, derived from radiographic measurements of the femoral artery; the real artery does not taper in such exponential fashion. In any case the overall taper of this vessel is small.

A pressure-diameter curve taken from Milnor and Bertram (1978) was utilized with dimensional relations to approximate this effect as a function of $z$ in the tapered vessel. Input pressure and flow pulses and other data were measured by the present authors. These pulses are illustrated in Figure 6 by solid lines.
Table 2  Parameter Values for Nonlinear Femoral Model and its Linearized Version

<table>
<thead>
<tr>
<th>System parameter</th>
<th>Femoral model</th>
<th>Linear model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluid density (μ, g/ml)</td>
<td>1.055</td>
<td>1.055</td>
</tr>
<tr>
<td>Fluid viscosity (μ, poise)</td>
<td>0.032</td>
<td>0.032</td>
</tr>
<tr>
<td>Cycle period (τ, sec)</td>
<td>0.576</td>
<td>0.576</td>
</tr>
<tr>
<td>Vessel length (L, cm)</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Taper constant (k)</td>
<td>0.02</td>
<td>0</td>
</tr>
<tr>
<td>Inner radius (r_i, cm)</td>
<td>0.23 exp(-kz)</td>
<td>0.22</td>
</tr>
<tr>
<td>Compliance (dn/dp, cm/mm Hg)</td>
<td>0.0019 exp(-kz) (p)^{-1/2}</td>
<td>1.5 x 10^{-4}</td>
</tr>
</tbody>
</table>

Characteristics of the models include, for the femoral model: inclusions of nonlinear wall compliance (a function of pressure and z), the radius a function of z, and the terms μ∂V_r/∂z, ρV_r(∂V_r/∂z), and ρV_r(∂V_r/∂r) in the equation of motion (V_r, axial velocity; V_z, radial velocity; r, radial coordinate); for its linear correlate: compliance independent of pressure and z, radius not a function of z, and omission of the above terms in the equation of motion.

Both models, as well as the input waveforms, are approximations to reality. Ideally, the input waveforms should represent antegrade pulses, free of reflection. However, they were drawn from a system with such contributions. Consequently, the harmonic content and model responses may vary. With such constraints in mind, we examined the kind and degree of alteration which may be imposed by the two models.

Table 3 lists, for each vessel type, the contribution to the total pressure loss, averaged over a cycle, for each term in the Navier-Stokes equation (for V_r) at the downstream plane (z = 10 cm). These terms are exclusive of the viscous properties of the wall. It should be noted that convective acceleration terms contributed little. This is so because wall compliance largely overcame the effect of small geometric taper.

Figure 6 illustrates upstream and downstream (10 cm) flow and pressure curves and associated Fourier series moduli for both the linear correlate and femoral artery models incorporating all losses. In these figures the upstream curves (solid lines and bars) are the inputs and the downstream curves (dotted lines and shaded bars) result from the calculations. Comparable alteration in flow waveform and phase has appeared elsewhere as a consequence of different nonlinear analyses (Barnard et al., 1966; Ling et al., 1973; Melbin and Gopalakrishnan, 1978). Upstream and downstream flow curves, obtained with cuff-type flow probes in the femoral artery (Milnor and Bertram, 1978) placed 3.7–5.3 cm apart, are not so greatly varied and appear more like linearly transmitted pulses. Differences relating to short distances, wall constraint, model properties, reflections, etc. make other than superficial comparison anecdotal at this time.

Figure 3b illustrates the transmission/10 cm obtained for a variety of models. The dash-dot curve is the transmission/10 cm expected as a result of only blood viscosity (Jager et al., 1965). The dotted line depicts the losses in our linear correlate due to wall viscosity alone. The slash curve is transmis-

![Figure 6 Pressures, flows, and the Fourier harmonics for pressure. Solid lines: upstream pressures and flows; solid bars: upstream pressure; dotted lines: downstream pressures and flows; shaded bars: downstream pressure. a: computations based on linear theory and linearized physical model; b: computations utilizing nonlinear theory and nonlinear physical model parameters.](image_url)

### Table 3  Contribution of Individual Navier-Stokes Terms to Pressure Change over 10 cm

<table>
<thead>
<tr>
<th>Navier-Stokes term</th>
<th>Femoral model (mm Hg)</th>
<th>Linear model (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Viscous</td>
<td></td>
<td></td>
</tr>
<tr>
<td>µ ∂V_r/∂z</td>
<td>−0.553 (67.3)</td>
<td>−0.443 (71.9)</td>
</tr>
<tr>
<td>µ (1/τ)∂V_z/∂τ</td>
<td>−0.169 (20.6)</td>
<td>−0.136 (22.1)</td>
</tr>
<tr>
<td>µ ∂V_r/∂r</td>
<td>0.000 (0.00)</td>
<td>0</td>
</tr>
<tr>
<td>Inertial</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ρ ∂V_r/∂t</td>
<td>0.033 (4)</td>
<td>−0.037 (6.1)</td>
</tr>
<tr>
<td>ρ V_r∂V_z/∂t</td>
<td>−0.046 (5.6)</td>
<td>0</td>
</tr>
<tr>
<td>ρ V_r∂V_r/∂r</td>
<td>−0.02 (2.4)</td>
<td>0</td>
</tr>
</tbody>
</table>
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Euler L (1862) Principia pro motu sanguinis per arterias determinando (1775). Opera posthuma mathematica et physica, Petropoli: Eggers 2: 814-823


Melbin J, Li K-J, Noordergraaf A (in press) Pulse propagation to vascular beds: Contribution of nonlinearities and system parameters, Cardiovasc Diseases


Preservation of Platelets during Extracorporeal Circulation in Sheep
A Comparison between Aspirin and Sulfinpyrazone

E.R. TOWNSEND, J. DUFFIN, M. ALI, J.W.D. McDoNALD, J.J. THIESSON,
J. MASTERSO N, P. KLEMENT, AND J.D. COOPER

SUMMARY We studied the effect of aspirin and sulfinpyrazone on changes in platelet count, thromboxane B₂ production, and pulmonary vascular resistance following the onset of veno-venous membrane oxygenator perfusion in sheep. Perfusion under identical circumstances was performed in three groups of animals, with one group serving as controls, one group receiving pretreatment with 1.5 grams of sulfinpyrazone iv, and one group receiving pretreatment with aspirin 50 mg/kg, iv. Both aspirin and sulfinpyrazone eliminated the rise in thromboxane B₂ and the increase in pulmonary vascular resistance seen in control animals. Platelet preservation was improved significantly with sulfinpyrazone pretreatment, but not affected by aspirin. We conclude that sulfinpyrazone and aspirin have different mechanisms of action as far as platelet preservation during extracorporeal perfusion is concerned. The major difference may be sulfinpyrazone's ability to reduce platelet adhesion to the membrane surface. Since both aspirin and sulfinpyrazone eliminated the pulmonary vascular response, but only sulfinpyrazone preserved platelets, it is apparent that the pulmonary vascular response is more closely related to thromboxane synthesis than to platelet disappearance. Circ Res 49: 452–457, 1981

BOTH aspirin and sulfinpyrazone have been utilized as antithrombotic agents and have been evaluated in recent clinical trials for the prevention of stroke (Canadian Co-operation Stroke Study Group, 1978) and myocardial infarction (Anturane Reinfarction Trial Research Group, 1980). The mechanism of action whereby these two drugs exert their antithrombotic activity is incompletely under-
Pulse wave propagation.
J K Li, J Melbin, R A Riffle and A Noordergraaf

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