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Computational Fluid Dynamics Modeling Techniques Using Finite Element Methods to Predict Arterial Blood Flow

8.1 Introduction

In the past, the study of hemodynamics has been approached from many different directions, by researchers from disciplines as diverse as medicine and engineering. In attempting to match the knowledge of fluid dynamics to the clinical reports of the patterns of arterial disease, researchers have discovered insufficiencies on both sides. This study identifies a methodology to address some unanswered questions. The broad objective of this hemodynamic study is to develop the capability for numerical prediction.

This investigation presents in vivo measurements of flow quantities and subsequent numerical flow and pressure calculation and validation, using measured flow parameters, vessel dimensions, and blood viscosity in a mildly tapered femoral artery of a living dog. While such a combined experimental–calculation approach may appear straightforward in principle, it is fraught with concerns about the reliability of in vivo measurements and the appropriateness of the method of flow calculation with regard to the non-Newtonian viscosity of blood, wall and inlet boundary conditions, and handling of the non-linear
convective acceleration terms and coupling of the pressure and velocity fields during the cardiac cycle. Moreover, giving statistical significance to such an approach would generally require numerous experiments and theoretical comparisons — a procedure which is beyond the scope of most detailed flow investigations such as this one.

Our study is modest and more along the lines of a bioengineering approach. We posed the question: What is the most accurate procedure for numerically calculating and validating arterial flows using in vivo measurements? In order to answer the question realistically, we identified two possible approaches and posed subsequent questions:

- What level of approximation is required to adequately describe the measured variation of pressure change with time, $\Delta p(t)$ and time-averaged pressure drop $\Delta p_a$, across a mildly tapered femoral artery segment of a dog given the measured velocity waveform $u(t)$?
- What level of approximation is required to adequately describe the measured variation of velocity with time, $u(t)$, and time-averaged velocity, $u_a$, given the measured pressure change with time, $\Delta p(t)$?

Clearly, the two approaches above are diametrically opposed, but the answer to either question would lead to the objective of this study. This question requires accurate measurements of $\Delta p(t)$, $u(t)$, and, thus, $\Delta p$ vs. $u$, which is of interest in and of itself for physiological flow and is rarely measured or reported in the literature. In the flow calculations, blood viscosity was assumed to be dependent upon shear rate as prescribed through the second invariant of the rate of strain tensor which, in turn, results in relatively unimportant elastic and time relaxation effects. This is believed to be a reasonable approximation for blood flow through arteries the size of the femoral artery in a dog.

On the other hand, since the arterial wall is, in general, very complex, being anisotropic (elastin, collagen, smooth muscle), viscoelastic (creep, stress relaxation, hysteresis), and under a mean and fluctuating state of three-dimensional stresses, it is very difficult to specify its dynamic behavior with certainty in computational methods. Clearly, the simplest assumption from a calculation point of view is to neglect wall interaction and motion, which is known to be relatively small, carry out the flow calculation for an assumed rigid wall, and then compare the calculations to in vivo flow data to appraise the extent to which the flow and pressure fields can be described. This is the approach used in this investigation. It is believed to provide some insight into arterial hemodynamics and flow calculation assumptions, albeit for a single vessel of specific size, shape, segment length, and particular pressure and velocity waveforms. Also, some implications with regard to in vitro arterial vessel model flow studies are inferred from this study since it provides an in vivo reference datum for construction of models with similar $\Delta p$ and $u$ characteristics during the cardiac cycle.

### 8.2 Background and Literature Review

In order to achieve the above-mentioned objectives, various issues must first be addressed. For any study in arterial blood flow dynamics, the following important issues require review:

1. The behavior of blood, the principle fluid;
2. The physiological flow regime, expressed in terms of the Reynolds number, the Womersley number, and the Dean number (for flow in curved geometry); and
3. The characteristics of blood ejection from the heart, which is pulsatile (unsteady) in nature.

First, we must consider the nature of the principal fluid, blood, itself. In large arteries (and in smaller ones like the femoral artery) which originate from the heart, blood, for most practical purposes, behaves as a homogeneous and continuous fluid medium, with characteristics similar to a Newtonian fluid, that is, a fluid in which the shear stress is linearly proportional to the rate of deformation. Further down the circulatory tree this assumption becomes increasingly less satisfactory, and by the time blood reaches the arterioles that feed the capillary bed, its rheology is complex. In flow through the capillaries, the concept
of a continuous fluid medium loses meaning, and the motion must instead be treated in terms of individual deformable erythrocytes (red blood cells) being transported by the surrounding plasma through narrow passageways sometimes smaller in diameter than the diameter of the erythrocyte itself in its relaxed state.

Second, when dealing with physiological applications, we must closely study the flow regimes, which are at least as complex and varied as the nature of the fluid itself. The character of a flow regime may be expressed, in most cases, in terms of its Reynolds number, a dimensionless parameter which measures the ratio of inertial force to viscous force acting on a fluid. In flows with a high Reynolds number, fluid inertia is the dominant force; in flows with a low Reynolds number, a viscous effect predominates. In the heart and aorta, the Reynolds number is high, approaching values for the transition (Re = 2000 ~ 3000) from a laminar to a turbulent flow. The Reynolds number decreases as the blood proceeds down the arterial tree; in the capillaries, the flow is in the so-called Stokes regime, where viscous forces overwhelm the inertial forces. Given its Reynolds number, the flow through most arteries such as the femoral artery is considered to be laminar.

A third factor requiring close attention is the pulsatile (unsteady) nature of blood flow from the heart. This adds to the complexity of the problem. Arterial vessels are often curved and branched, producing complicated secondary motions of blood and entry phenomena which are far more difficult to analyze than the fully developed flow in a straight tube of infinite length. A further complication is the distensibility of arterial vessels, which deform in a viscoelastic fashion under pressure variations associated with the pulsatility of the blood flow.

This study focuses only on flow behavior. However, many other arguments based on chemical processes, mass transfer, and various other unexplained, naturally occurring phenomena have been made. Although various authors have reported experimental and numerical procedures for obtaining the pressure–flow relationship, the numerical prediction of an in vivo pressure drop using the Navier–Stokes equations with the convective terms in an arterial geometry with physiological shape has not been documented in full.

Flow reversal in a dog's artery and phase lag between the pressure gradient and the flow have been reported by McDonald and his co-workers and have also been numerically studied by Womersley. A factor of only ± 1.8% radial dilatation Δrw/rw is reported for the resting iliac artery of a dog. Compliance of the resting femoral artery of a dog will be even smaller than the iliac artery. Ling et al. have reported that, for a dog aorta, flow profiles developed locally and transiently during the passage of the pressure-gradient wave. In vivo velocity and shear rate data have indicated relatively flat velocity profiles. Back et al. have measured in vitro pulsatile velocity and pressure in a human femoral artery model with a reverse lumen curvature. Talbot and Berger have illustrated the relationship of the acceleration and deceleration of velocity to the pressure distribution. For a steady flow analysis, Back et al. have measured flow resistance in models of a curved femoral artery of a human. They have predicted that in order to have a fully developed secondary flow, a relatively high Dean number and curved segment angle are required.

The measured non-Newtonian properties of blood, i.e., shear thinning and viscoelasticity, have been reported in the literature. Flow measurements in an atherosclerotic, curved, tapered femoral artery model of a human have been performed by Back et al. For a straight tapered model of the smooth femoral artery, the mean flow measurements have shown significant pressure drop which can be estimated from momentum considerations using the assumption of a local Poiseuille flow. For a mildly tapered femoral artery of a dog, the numerical study of Banerjee et al. has predicted oscillatory shear stress and a phase lag between pulsatile pressure drop and velocity.

Based on the questions posed earlier and discussion of possible approaches, this study was divided into two parts. Study 1 attempted to propose a method to calculate pressure changes in an artery of a dog using an arterial angiogram, the non-Newtonian viscosity of blood, and an instantaneous velocity measurement by non-invasive means. Study 2 attempted to propose an alternate method to calculate
velocities using instantaneous pressure gradient measurements. Clearly, either of the two studies validated experimental results with numerical methods.

### 8.3 Uncertainties in the Proposed Studies

#### Experimental Uncertainties

Rather than using *in vitro* measurements, these studies aimed to compare and validate the numerical calculations with *in vivo* measurements. It is to be noted that, in the past, *in vitro* measurements were generally found to be easier to compare with numerical calculations since it is comparatively simpler to control any *in vitro* experiment\(^*\) than to control an *in vivo* experiment. In view of this, Studies 1 and 2 were difficult exercises since they involved the greater uncertainties that accompany *in vivo* measurements.

Also, it is practically impossible to measure, and hence numerically verify, *in vivo* velocity profiles at any instant of time near the wall region. At any instant of time, the Doppler flow cuff measures a spatial average velocity only in the core region and excludes the near-wall region of an artery. However, through calibration, the spatial average velocity across the artery is determined. Nevertheless, this poses a difficulty in specifying temporal and spatial dependent velocity boundary conditions, as may be observed in Study 1. In contrast, the measurement of pressure drop is usually more accurate and reliable; hence, such a pressure boundary condition is easy to apply, as may be seen in Study 2. Clearly, from a calculational point of view, Study 1 has greater uncertainties than Study 2.

#### Numerical Uncertainties

It should be noted that the heart rate of a dog (128 beats/min) is one-and-a-half times faster than the heart rate of a human (75 beats/min), and, therefore, the rate of change of an instantaneous boundary condition over a pulse cycle is higher for a dog than for a human. In general, a numerical calculation for a dog artery has a more stringent condition than for a human artery.

For a numerical simulation of flow, particularly pulsatile flow, calculation of pressure is inherently difficult. Since pressure calculation involves second-order spatial derivatives and a first-order temporal derivative of velocity, any sharp variation in the inlet boundary conditions, as in the case of pulsatile flow, significantly affects the pressure calculation.

Since velocity is used as an inlet boundary condition, as in Study 1, an inherent mass balance is automatically achieved. Only the momentum balance needs to be performed in the flow domain. In contrast, when a pressure boundary condition is applied, as in Study 2, both mass and momentum balances need to be achieved. Clearly, from a numerical point of view, Study 2 is more difficult than Study 1.

### 8.4 Experimental Method

The animal experiments were performed in Dr. Crawford’s laboratory at the USC School of Medicine using instrumentation transported from the Jet Propulsion Laboratory (JPL) to USC with subsequent data reduction at JPL. The animals used were mongrel dogs which were unclaimed at local government pounds. They were certified by USC veterinarians for single experiment utilization, and the experimental protocol was approved by the USC Animal Experimental Board.

Methods for measuring *in vivo* static pressure changes along a segment of the femoral artery without inserting a catheter into the lumen, which can disturb the flow, were investigated initially. One way to take this measurement is by inserting a small plastic tube with a flared end through an incision in the artery wall. By suturing the artery around the tube with the flared end flush with the inner arterial wall surface, we are able to replicate a sidewall static pressure tap. This technique, however, has not proved successful in measuring the *in vivo* static pressure change along a length of an artery because the procedure causes local vaso-constriction and some thrombosis at the injured sites. Consequently, the narrowing of
the arterial cross-sectional area in the vicinity of the plastic tubes precludes obtaining meaningful measurements.

Another way to measure static pressure changes is to use small arterial branches as pressure taps. This involves severing a branch some distance from the main lumen, inserting a small plastic tube, and tying the tube externally to prevent blood leakage. Care must be exercised during the experiment to prevent clotting in the plastic tube by frequent flushing with heparin. This technique is used in the animal experiments, and details are reported here.

Animals were lightly anesthetized with ketamine–xylazine given intramuscularly, and a transcutaneous catheter was placed into a dorsal foreleg vein, maintained open with a heparin lock, for control in case vascular support or euthanasia became necessary. Under this light anesthesia, transthoracic intubation was performed, and anesthesia was controlled thereafter using inhalation nitrous oxide/methoxyflurane with 20% oxygen and assisted respiration. An angiography catheter was then introduced into a femoral artery and advanced to the aortic bifurcation for the injection of standard angiographic contrast material.

The opposite femoral artery was surgically exposed from 1 cm beyond the inguinal ligament to the adductor canal, and side (muscular) branches were identified. Most of these were ligated, but at appropriate distances some are cannulated for lateral wall pressures. An L and M Doppler flow meter is positioned just after the proximal pressure tap. Flow measurements, absolute and differential pressures, and angiograms are taken during resting and vasodilated flow (adenosine or the radiographic contrast agent renografin). Correlation of angiograms and physiological data is made by use of a radiation sensitive voltage producing device in the radiographic field. Just after euthanasia by a large injection of pentobarbital while the animals are still anesthetized, post-mortem casts are made of the vascular segment using a silicone rubber bolus. Care is taken to obtain mean in vivo arterial reference diameters by adjusting the perfusion pressure. Details on the measurements, instrumentation, and data acquisition system are given below.

**Technical Details, Instrumentation and Data Acquisition System**

For studying the femoral artery, contralateral femoral artery catheters were used to measure iliac bifurcation pressures with the tip positioned with a fluoroscope. The tip of the probe was closed and faced downstream. The static pressure holes were on the side. The transducer control instrumentation was part of the EforM in the USC laboratory. This transducer was calibrated with a mercury manometer.

Based on a pre-test angiogram, a section of the femoral artery was selected between two branches. Any intervening branches were tied off. The two end branches were isolated, cannulated with a static pressure tube, and each branch was sutured tightly around its tube. A fluoroscope was used to position the pressure tap tube normal to the artery with the tip at the wall location. The tube was then secured in position to the surrounding tissue. The animal’s leg was positioned so that the arterial section was level. The pressure tap tubes were connected to the two sides of the Validyne differential pressure transducer (model number DP103-20), which has a frequency response of at least 1000 Hz for incompressible fluid such as blood, and appropriate valving to permit frequent flushing and zero checking (Fig. 8.1). The transducer was filled with heparin solution used in frequent flushing to prevent clotting at the tips. 100 cm of water at the full scale diaphragm plate were used in the transducer to minimize the time delay of the response due to fluid movement as the transducer diaphragm deflected. A 200 Hz low-pass internal filter was used. The excitation voltage and calibration for the differential pressure transducer were determined at JPL under static and steady flow conditions with a water manometer. The instantaneous in vivo pressure difference signals were quite smooth.

The velocity measurement was made with an L and M Doppler flowmeter. Based on the pre-test angiogram, the position for the flow cuff in a relatively straight section of the artery was selected. The appropriate sized flow cuff was placed around the vessel. The skin incision flaps were pulled up and secured, forming a wall to hold the saline solution in which the flow cuff was immersed. For blood flow, an 8.2 MHz excitation signal provided a good output. Both the time-mean and instantaneous fluctuating
Signals were obtained with the unit. The fluctuating signal had a high noise level even with a steady flow in bench experiments at JPL. A 30 Hz internal low pass filter was used to obtain a localized 0.03 s average throughout the approximate 0.47 s heartbeat of a dog. Filtering below 30 Hz has been observed to reduce the level of the peaks. The instrument was calibrated at JPL for steady flow of blood through silicon tubing. These calibrations have been shown to be good for the mean of a fluctuating flow in bench tests at JPL.

Additional instrumentation (Fig. 8.1) from JPL provided for the amplification/attenuation and/or time averaging of the three fluctuating signals and their recording on a magnetic tape cassette in the ±1 volt range. A fourth channel was used for voice documentation during the tests.

Several angiograms of the arterial section were obtained during the experiments. The renografin solution injection for these angiograms caused vasodilation. Data was normally obtained during this process.

At a later date, the recorded data was reduced at JPL. The schematic for the instrumentation used to retrieve the tape recorded signals is given in Fig. 8.2. The smoothly varying differential pressure signal...
was used to provide a marker via a trip circuit and a pulse generator. This marker was used to excite the correlator and initiate a 256 point additive accumulation during the next window, which was typically 0.4 s. Generally, 80 to 160 such sums were averaged at each time t in the cardiac cycle. The resulting distribution was displayed on the oscilloscope, could be plotted on the X-Y plotter, and was sent in digital form at 110 baud to a computer for further processing.

Correlator settings and reference information such as the mean readings into and out of the tape recorder were also keyed into the computer via a teletype. Each signal on a given segment of tape was thus processed individually, but always relative to the timing of the differential pressure signal. The computer corrected for the calibrations of the various instruments used to process the signal and provided a plot of the data.

Interpretation of these results required knowledge of the physical topography of the vessel section and the blood properties. The former was obtained using the densitometric image scanning methods developed by the JPL Biomedical Image Processing Laboratory. Therefore, the lateral extent and the cross-sectional area along the vessel section could be obtained from the angiograms. Background interference could be corrected for by using a pre-angio x-ray. Direct visual comparisons could also be made with the silicone cast of the section.

The viscosity of the blood drawn from the animal was measured with a Brookfield cone and plate viscometer, which measures the shear stress at shear rates up to 450/s. These measurements were made at several temperatures including the in vivo temperature with a chemically stabilized sample stored under refrigeration. The density was obtained by weighing 10 cc of this blood sample.

**Arterial Geometry**

A tracing of the x-ray of a portion of the femoral artery of a dog where the measurements are taken is shown in Fig. 8.3. The pressure drop across the segment is measured by using two small branch arteries.
which are ligated and connected by tubing to a Validyne transducer. The cuff for the Doppler flowmeter is located near the first branch, as shown in Fig. 8.3.

For Studies 1 and 2, the vessel segment (Fig. 8.3) is simplified and kept relatively straight with mild taper. The vessel diameter at the first branch tap (referred to as port 1) is \( d_1 = 3.8 \text{ mm} \), and at the second branch tap (referred to as port 2) is \( d_2 = 3.6 \text{ mm} \). The axial distance between the branch pressure taps is 52 mm, so that the ratio of axial distance and diameter \( d_1 \) of the vessel segment is 13.7.

**Formulation**

The objective of this numerical study was to obtain the time-dependent solution of an incompressible, non-Newtonian fluid for the selected geometry (Fig. 8.3). The flow is described by the conservation equations of fluid mass and momentum. A finite element method (FEM) was used to solve the two conservation equations and, therefore, to obtain the velocity, wall shear stress, and pressure distributions. These two equations are presented as follows:

\[
\rho \left[ \frac{\partial u_i}{\partial t} + u_j u_{i,j} \right] = \sigma_{ij,j} + \rho f_i \tag{8.2}
\]

where \( i, j = 1, 2 \) for axisymmetric flows, \( u_i \) is the \( i \)th component of the velocity vector, \( \rho \) is density, \( \sigma_{ij} \) is stress tensor, and \( f_i \) is the body force. Furthermore,

\[
\sigma_{ij} = -\rho \delta_{ij} + \tau_{ij} \tag{8.3}
\]

\[
\tau_{ij} = 2\eta \varepsilon_{ij} \tag{8.4}
\]

\[
\varepsilon_{ij} = 0.5 \left( u_{i,j} + u_{j,i} \right) \tag{8.5}
\]

Here, \( p \) is the pressure, \( \tau_{ij} \) is the deviatoric stress tensor, \( \varepsilon_{ij} \) is the shear rate tensor, \( \eta \) is the tensor viscosity, and \( \delta_{ij} \) is the Kronecker delta. This study was conducted using the axisymmetric coordinate system with symmetry about the z-axis, i.e., all field quantities were independent of \( \theta \), and the circumferential component of velocity \( u_\theta \) was zero. Axial components of flow were reported as a function of \( r \) along the z-direction.

The stress vector \( s_i \) at a point on the boundary of a fluid element is defined by

\[
s_i = \sigma_{ij} n_j \tag{8.6}
\]
For a known element and the solution field, the stress component $s_i$ on the boundary at the Gaussian integration points was evaluated. Subsequently, the normal and tangential components of stress vectors were obtained after applying the appropriate transformations.

The Galerkin formulation using finite elements was applied here in order to discretize the above continuity and momentum equations; this resulted in a set of non-linear algebraic equations of the form

$$M\dot{V} + K(U)V = F(U), \quad (8.7)$$

where $U = (u_1 \ u_2)$, $V = (u_1 \ u_2 \ P)$, $K(U)$ is the global system matrix developed from the momentum balance, $M$ is the mass matrix, $u_i$ is the velocity unknown, and $F$ is the forcing function (including body forces and boundary conditions). Four nodal quadrilateral elements were considered for this study. The mesh plot for the artery is shown in Fig. 8.4.

FIGURE 8.4 The mesh plot of the femoral artery shown in Fig. 8.3. For Studies 1 and 2 the vessel segment is simplified and kept relatively straight with mild taper.

In order to numerically solve the momentum and continuity equations for velocity and pressure fields, various solution techniques were used. In a FEM, solution techniques include mixed and penalty formulations. For the mixed formulation, both velocity and pressure in the momentum and continuity equations are independent variables and were solved simultaneously, whereas for the penalty formulation, only velocity is an independent variable, and pressure was approximated by the dilation rate of liquid. In other words, the FEM was not directly applied to the system of equations but rather to a perturbed system of equations in which the continuity requirement was weakened by a penalty parameter, $\varepsilon$. The pressure was approximated as follows:

$$u_{ij} = -\varepsilon p_i, \text{ where } \varepsilon = 1 \times 10^{-9} \quad (8.8)$$

For high aspect ratios of the elements, a small penalty parameter is recommended. Physically, this can be equated to simulating a flow that has an insignificant compressibility effect. This approach has the advantage of eliminating one of the dependent variables $p^*$, which is then recovered by post-processing from the velocity field by

$$p^* = -u_{ij}^{**} / \varepsilon \quad (8.9)$$

Clearly, both the pressure and velocity fields must be determined in the calculation method. Considering the merits and demerits of numerical schemes and associated inlet boundary conditions, a penalty formulation was used for Study 1, whereas a mixed formulation was chosen for Study 2.

The matrix equation (8.7), representing a discrete analog of the original equations for an individual fluid element, was constructed, assembled, and solved. For spatial integration, the number of iteration steps was limited to ten at each time step with a combination of the successive substitution and quasi-Newtonian scheme.
The numerical simulation of a pulsatile flow required a time integration method. The implicit time integration scheme used in this study was the second-order trapezoidal method with a variable time step, which is dependent on the magnitude of temporal inlet velocity and its gradient change. Depending on the physiological velocity pulse shape, the time steps were varied between $1 \times 10^{-4}$ to $1 \times 10^{-5}$ s. The finite-element computer code was used to formulate and solve this matrix equation. For the present study, a Sun Ultrasparc 2 with a speed of 200 MHz, 256 MB RAM, and a 4 GB disk was used and the post-processed results are downloaded to an IBM-PC computer for plotting the results.

In comparison to the core elements of the artery, the element sizes near the wall were kept small in order to achieve accuracy for flow parameters. The aspect ratio of the elements was chosen to be less than 10. For validation of the numerical computation, two separate computer modeling runs were performed at peak systolic flow with different convergence criteria as follows: both the relative velocity error with respect to the previous step and the relative residue error compared to the initial value were set to be 2% and 1%. Furthermore, the overall convergence was confirmed by increasing the total number of meshes by 20% over that of the previous run, and the two results were compared to check for accuracy. When the improvement with 20% more meshes was less than 1% in velocity vectors, wall shear stress, and pressure, the computation was considered to be accurate. In addition, the available experimental data and the numerical prediction of pressure drop were compared. The analysis of results is from the computation with the least CPU time, i.e., with less than 2% for both relative velocity error and relative residue error. The CPU time for each time step is approximately 4.38 s for Study 1 and 2.86 s for Study 2.

### Boundary Conditions

Study 1 was conducted with a pulsed velocity inlet condition whereas a pressure gradient was used in Study 2. The inlet conditions used in both studies were obtained from in vivo experimental data. The outflow boundary conditions did not need to be specified at the exit since its values were effectively determined by extrapolation similar to the finite difference schemes. A no-slip boundary condition was specified on the rigid arterial wall. Since the artery was relatively straight, the flow domain was modeled as axi-symmetric. Specific details of boundary conditions for each study are provided below.

#### Boundary Conditions for Study 1

Obtaining an accurate in vivo instantaneous velocity inlet profile with a non-invasive method is difficult; such an instantaneous velocity profile could have a spatial velocity distribution that is either parabolic, uniform, or any combination of the two. Given this experimental difficulty and uncertainty, the measured core velocity is the most accurate and must be considered. Since the femoral segment is deep within the circulation and is branch-free upstream, the numerical calculations were performed for the instantaneous velocity and a parabolic spatial inlet flow condition for which $u_{cl}$ is twice the calibrated spatial mean velocity $u_c$. Fig. 8.5a shows the calibrated in vivo spatial mean velocity, as measured by an ultrasound Doppler flow cuff. Further details are provided in the results section below.

The inlet core velocity $u_{cl}$ obtained by curve fitting is shown in Fig. 8.6a, and used as an input for the present numerical simulation. The calculations are started at an axial distance of 2 cm upstream of port 1 for calculational stability purposes. The selected time steps for flow analysis are marked from 1 to 8 on the pulse.

#### Boundary Conditions for Study 2

Measured pulsed pressure drop was applied at the inlet section where port 1 is located (Fig. 8.6b). Since there was hardly any arterial curvature, the pressure variation in the radial direction was kept constant. Based on the applied pressure drop at the inlet, the flow field developed in the flow domain. For Study 2, since pressure drop was the boundary condition, the flow domain was truncated at port 1 and port 1 locations.

Determination of multi-dimensional arterial flow through viscoelastic vessels is beyond the scope of current computational capability. In the literature, physiological wall motion data for a dog is available which may be useful in developing simpler interactive computer codes.
FIGURE 8.5  Doppler flow velocity, pressure drop, and pressure measurements in the femoral artery of a dog. The dashed curves are mean values. The in vivo spatial mean velocity $u_i$ in the femoral artery along a pulse cycle was obtained by a calibrated Doppler flow cuff (Fig. 8.5a). Arterial pressure drop and pressure are shown in Fig. 8.5b and Fig. 8.5c, respectively.
In order to calculate the shear rate-dependent non-Newtonian viscosity in the flow field, the local shear rate, $\dot{\gamma}$, was calculated from the velocity gradient through the second invariant of the rate of strain tensor, $\mathbf{II}$, as follows:

$$\dot{\gamma} = \sqrt{\frac{1}{2} \mathbf{II}} = \sqrt{\frac{1}{2} \left[ \sum_i \sum_j \dot{\gamma}_i \dot{\gamma}_j \right]}$$  \hspace{1cm} (8.10)

After the local viscosity is determined by the blood model, Eq. 8.11, the local shear stress, $\tau (= \eta \dot{\gamma})$, is calculated. Schneck's best three variable model (B3VM)\textsuperscript{20} was used in our earlier calculations. However, during the course of Studies 1 and 2, a more accurate blood model, namely the Carreau model,\textsuperscript{10} was used.

Non-Newtonian Blood Viscosity for Studies 1 and 2

The Carreau model was used to represent the shear rate-dependent non-Newtonian blood viscosity whose model constants were obtained by a curve fitting of available shear rate-dependent blood viscosity data in the literature.\textsuperscript{10}
where $\lambda$ (characteristics time) = 3.313 s, $n = 0.3568$, $\eta_0 = 0.56$ poise, and $\eta_\infty = 0.0345$ poise. The dimensionless frequency parameter ($\alpha = 0.5 \sqrt{\frac{\omega}{\nu}}$) calculated based on infinite shear rate viscosity for a dog was 3.7.

8.5 Results

Animal Hemodynamic Data

The Doppler blood flow velocity measurements and pressure drop measurements are shown in Fig. 8.5 as a function of time in the same plot. From these two measurements, $\Delta p$ vs. $u$ is plotted to show hysteresis effects (Fig. 8.7).

The flow signal was tri-phasic with a brief period of reverse flow during the early part of diastole. At peak systole, the largest instantaneous pressure drop of $-4.5$ mm Hg was measured. The largest instantaneous pressure rise of $+2.7$ mm Hg occurred during flow reversal. During both of these peak conditions, there was little phase lag as is evident in Fig. 8.5.

To help identify the loop type hysteresis curve (Fig. 8.7), points labeled A through H are shown along the velocity wave form in Fig. 8.5 for a cardiac cycle beginning with the systolic phase (point A). During blood flow acceleration to peak flow, points A, B, and C lay along the upper branch of the hysteresis curve (Fig. 8.7), while during the flow deceleration phase from peak to reverse flow, points C, D, and E lay along the lower branch. For the second flow acceleration phase during diastole, points E, F, and G traverse from the lower branch of the hysteresis curve to the upper branch vicinity. During the latter part
of diastole where flow deceleration occurs again, points G, H, and A loop above the upper branch of the hysteresis curve to begin again the next cardiac cycle which is also shown in Fig. 8.7.

In addition to the pulsatile flow data, there is also interest in the mean flow data. The resting heart rate of a dog is 128 beats/min and therefore, the period, T, of a heart beat is 0.47 s. Using a digital voltmeter as shown in the previous section, the time-averaged blood flow velocity, \( u_a = 15.1 \text{ cm/s} \), and time-averaged pressure drop, \( \Delta p_a = -0.59 \text{ mm Hg} \), were obtained and are shown in Fig. 8.5 by the dashed curves. In this case, the ratio of peak pressure drop at peak velocity to the mean value, \( \Delta p_p/\Delta p_a \), was 7.6, thus indicating the relatively large variations in instantaneous pressure change compared to the mean value, including during the flow reversal phase. The corresponding value of the ratio of peak flow velocity to the mean value, \( u_p/u_a \), was 3.3, a value which is less than half of the pressure drop ratio, \( \Delta p_p/\Delta p_a \). A more complicated pressure change–flow relationship relative to the mean flow value was evident during diastole.

To place the hemodynamic data in perspective, some comments are in order. Measurement of the viscosity of the dog’s blood at the \( \text{in vivo} \) temperature gave \( \eta = 0.037 \text{ poise} \), and the measured blood density, \( \rho \), was equal to \( 1.04 \text{ g/cm}^3 \). The time-averaged flow rate \( Q_a (= u_a A_1) \) based on the upstream vessel diameter \( d_1 (3.8 \text{ mm}) \) was \( 102 \text{ ml/min} \). Since the mean flow Reynolds number \( Re_a (= 4 Q_a/\pi \nu d_1) \) was only 161, the flow was expected to be laminar. Considering the mean diameter \( \bar{d} = 3.7 \text{ mm} \), the time-averaged value of the pressure drop \( \Delta p_a \) using the Poiseuille relation

\[
\Delta p_a = -128\eta Q_a/\pi \bar{d}^4
\]

was \( -0.54 \text{ mm Hg} \). A further correction for mildly uniform taper was made from the momentum consideration; this then gave a value of \( \Delta p_a = -0.57 \text{ mm Hg} \). Since the measured value of \( \Delta p_a \) was only about 3% higher, this afforded a reasonable estimate of the mean pressure drop and instilled confidence in the accuracy of the measurement technique. Also considering the mean diameter \( \bar{d} = 3.7 \text{ mm} \), the time-averaged value of the wall shear stress \( \tau_{wa} \) using the Poiseuille relation

\[
\langle \tau_{wa} \rangle = 32\eta Q_a/\pi \bar{d}^5
\]

was 12.7 dynes/cm².

Finally, although the dimensionless frequency parameter, \( \alpha = 0.5 d_1\sqrt{\nu/\omega} \) was 3.7 and hysteresis effects were evident during the flow acceleration and deceleration phases of the cardiac cycle, phase variations between the velocity and pressure difference signals were relatively small at peak systole.

With the experimental data at hand, we turned to the task of numerically calculating the flow field and pressure differences by solving the momentum conservation equations, including the shear rate-dependent viscosity of blood.

**Flow Calculation Description**

This section has been divided in two parts: the results of Study 1 are followed by the results of Study 2. This section clearly demonstrates the agreement and disagreement between experimental measurement and numerical calculation. The calculations in Study 1 for a parabolic inlet profile were consistent with the Doppler flowmeter calibration where core flow velocities were about twice the spatial mean velocities shown in Fig. 8.5, unlike the earlier calculations which corresponded to assumed lower centerline velocities, which were incorrectly interpreted from the Doppler calibration, by a factor of about two. Also, these calculations used a more accurate Carreau model for shear rate-dependent non-Newtonian blood viscosity (\( \eta \)) than the Wilburn and Schneck model used in the earlier calculations.

**Flow Calculation for Study 1**

Temporal and spatial variations of the velocity profile were obtained along the radial directions of ports 1 and 2, whereas shear rate and non-Newtonian viscosity were reported at each port location. Shear
stress was calculated along the arterial wall between ports 1 and 2. In vivo pressure drop measurements across the ligated arteries (ports 1 and 2), which were connected to the tubing leading to a pressure transducer, were reported along with the validation of numerical pressure drops between ports 1 and 2.

This section presents detailed flow analysis for parabolic inlet velocity profiles. Results for the systolic acceleration (Fig. 8.6a) are represented by time steps 1 and 2, whereas results for systolic deceleration are represented by time steps 3 and 4. Time steps 5 and 6 represent an accelerating negative flow and a decelerating negative flow for early diastole, respectively. Time steps 7 and 8 represent an accelerating and decelerating positive flow for late diastole, respectively. Fig. 8.6a indicates the exact time that corresponds to the number of the time step; the figure shows that the velocity at the inlet is positive for time steps 1-4, 7, and 8, and negative for 5 and 6.

**Velocity Distribution for Parabolic Inlet Velocity Profile**

Fig. 8.8a shows the spatial distribution of the axial velocity profile for different time steps at port 1. The instantaneous velocity profiles of curves 1–4 are positive, with a centerline velocity that rises to 93 cm/s near peak systole; for curve 5, a flow reversal with velocity −7.2 cm/s is obtained near the wall, although the core velocity is still positive with a value of 9.7 cm/s. In contrast, the velocity for curve 6 is less negative with a maximum value of −2 cm/s near a distance halfway along the radius. For late diastolic flow, curves 7 and 8 show similar velocity profiles with a lower magnitude than that observed during systolic flow. Flow reversal near the wall during the late decelerating flow with a positive core velocity is an interesting phenomenon. Since the calculations were initiated upstream of port 1, some flow development occurred before port 1 during the cardiac cycle. For example, during flow acceleration phases (curves 2 and 7) the core velocity profiles became flatter than the inlet profiles, and during the reverse flow phase (curves 5 and 6) the velocity profiles became M-shaped as noted.

Similar trends were observed at port 2 (Fig. 8.8b) with variations of velocity caused by the mild taper. Due to the gradual taper of the artery, a higher core velocity of 51 cm/s was obtained at port 2 (compared to 45 cm/s at port 1) for curve 2 during the accelerating phase of the systolic flow. This trend was partially reversed during the decelerating phase of the systolic flow. At port 2, the centerline velocity for curves 3 and 4 were 90 cm/s and 70 cm/s, respectively, whereas at port 1 the values were 93 cm/s and 69 cm/s, respectively. Furthermore, in comparison to port 1, curve 5 at port 2 showed a slightly higher value for the reversed peak flow with a value of −7.6 cm/s. The velocity for curve 6 had a maximum value of −2.1 cm/s.

**Shear Rate for Parabolic Inlet Velocity Profile**

For a single cardiac pulse with a parabolic velocity profile, an oscillating wall shear may be observed in Figs. 8.9a and b. Spatial variations in the shear rate for selected time steps 1-8 (Fig. 8.6a) are shown in Fig. 8.9a (port 1) and 8.9b (port 2) with arrow marks indicating the oscillations at the wall regions. The magnitude of the wall shear rate at each time step is larger at port 2 than at port No. 1, indicating the effect of the taper.

Fig. 8.9c shows the complete time history for the entire pulse cycle. At port 1, the shear rate varied from zero to a maximum value of 1350 s⁻¹ at t = 0.089 s (before peak systole, between time steps 2 and 3 of Fig. 8.9) and attained a zero shear rate during the decelerating phase of late systole (between time steps 4 and 5). It then reached a maximum negative shear rate of −310 s⁻¹ at t = 0.225 s (early diastole, near the peak negative velocity after time step 5) which was followed by a shear rate of 494 s⁻¹ at t = 0.296 s (before the late diastolic peak positive flow, after time step 7). At port 2 (Fig. 8.9c) the shear rate varied from zero to a maximum positive value of 1555 s⁻¹ at t = 0.089 s and attained a zero shear rate value during the decelerating phase of late systole, before reaching a minimum shear rate of −340 s⁻¹ at t = 0.225 s, which was followed by a shear rate of 565 s⁻¹ at t = 0.296 s before the peak diastolic positive flow.

The steeper slope in the curve of the wall shear rate vs. time at port 2 compared to port 1 (Fig. 8.9c) was due to the taper during both acceleration and deceleration. This in turn affected the non-Newtonian viscosity, the shear stress, and the pressure drop. The systolic decelerating slope of the shear rate at port 2 was greater than at port 1, causing the shear rate lines (Fig. 8.9c) to intersect after time step 4 at
At port 2, the magnitude of the extremum values of the wall shear rate was larger than that for port 1. This can be attributed to the arterial taper.

**Non-Newtonian Viscosity of Blood**

The non-Newtonian viscosity of blood was modeled using whole blood. The non-Newtonian blood viscosity at the wall is presented in Fig. 8.10 for the parabolic inlet flow. It is apparent that for high shear, the non-Newtonian viscosity is low and vice-versa. In Fig. 8.9c, the shear rate at port 2 is higher than that at port 1, resulting in a lower non-Newtonian viscosity. The viscosity during systole, in general, is smaller than that during diastole. At peak systole in Fig. 8.10, the non-Newtonian viscosity is 0.037 poise (1 poise = 0.1 Pa/s) which is the infinite shear rate viscosity measured using dog blood in a cone and plate viscometer. Near the zero shear rate, the non-Newtonian viscosity is 0.16 poise, which is approximately four times the infinite shear rate viscosity. The non-Newtonian viscosity oscillates during the decelerating part of systole which is due to the oscillation in shear rate between positive and negative values.

Due to the higher systolic decelerating slope for the parabolic inlet condition (Fig. 8.9c), the shear rate at port 2 at the end of systole and at the beginning of diastole was lower (larger negative value) than at port 1, which caused a locally lower value of the non-Newtonian viscosity (Fig. 8.10).

FIGURE 8.8 Radial velocity distribution, with a parabolic spatial inlet condition, for different time steps along a pulse cycle at port 1 (Fig. 8.8a) and port 2 (Fig. 8.8b).
FIGURE 8.9a-b  Radial shear rate distribution, with a parabolic spatial inlet condition, for different time steps along a pulse cycle at port 1 (Fig. 8.9a) and port 2 (Fig. 8.9b).

FIGURE 8.9c  Temporal variation of wall shear rate distribution at port 1 and port 2 for a parabolic spatial inlet condition.
Fig. 8.11 shows the instantaneous shear stress along the arterial wall for parabolic inlet conditions. The shear stress at the wall illustrates the combined effect of instantaneous wall shear rate and local non-Newtonian viscosity.

During the systolic acceleration at port 2, the wall shear stress shown in Fig. 8.11 increases from zero to more than 47 dynes/cm², followed by a drop to a value of less than −13 dynes/cm² during early diastole (time step 5). Subsequently, during the late diastolic phase (time step 7), the shear stress rises to a positive value of more than 22 dynes/cm² followed by a reduction to zero at the end of a pulse cycle. The maximum value of the wall shear stress is 57.0 dyn/cm² at t = 0.089 s (before peak systole between time steps 2 and 3). As observed in Fig. 8.11, the effect of the taper is indicated by a gradual rise in the magnitude of shear stress with axial distance for all the time steps.

FIGURE 8.10 Temporal variation of non-Newtonian viscosity at the wall for port 1 and port 2 for parabolic spatial inlet conditions.

Shear Stress

Fig. 8.11 shows the instantaneous shear stress along the arterial wall for parabolic inlet conditions. The shear stress at the wall illustrates the combined effect of instantaneous wall shear rate and local non-Newtonian viscosity.

During the systolic acceleration at port 2, the wall shear stress shown in Fig. 8.11 increases from zero to more than 47 dynes/cm², followed by a drop to a value of less than −13 dynes/cm² during early diastole (time step 5). Subsequently, during the late diastolic phase (time step 7), the shear stress rises to a positive value of more than 22 dynes/cm² followed by a reduction to zero at the end of a pulse cycle. The maximum value of the wall shear stress is 57.0 dyn/cm² at t = 0.089 s (before peak systole between time steps 2 and 3). As observed in Fig. 8.11, the effect of the taper is indicated by a gradual rise in the magnitude of shear stress with axial distance for all the time steps.

FIGURE 8.11 Wall shear stress distribution for different time steps along a pulse cycle between ports 1 and port 2 for parabolic spatial inlet conditions.
Pressure Drop

Since Banerjee et al.\textsuperscript{6} reported pressure drop results calculated for different discontinuous pressure formulations (e.g., bilinear, linear with local basis function, and linear with global basis function), only the results obtained using the discontinuous bilinear pressure formulation\textsuperscript{5} are presented here for the parabolic inlet condition. The pressure drop calculated for different pressure formulations is presented in Banerjee et al.\textsuperscript{6} Curve 1 in Fig. 8.12 represents the pressure drop for the parabolic inlet condition. Curve 2 represents the experimentally obtained \textit{in vivo} pressure drop measurements.

For the parabolic inlet condition, the calculated pressure drop has a maximum value of –6.7 mm Hg at \( t = 0.087 \) s (represented by the upward spike) during peak systole and reaches 3.1 mm Hg at \( t = 0.192 \) s during early diastole (represented by the downward spike). Experimentally measured peak pressure drops are –4.5 mm Hg at \( t = 0.110 \) s during systole and 2.7 mm of Hg at \( t = 0.225 \) s during the early diastolic phase of flow. For mid-diastole, the second peak pressure drop is also overestimated (–3.0 compared to –1.7 mm Hg) and in late diastole, a pressure rise was calculated but a pressure drop was measured. Numerical calculation of the average pressure drop is –0.645 mm Hg for the parabolic inlet condition, whereas experimental data show a value of –0.59 mm Hg. The 10% difference between the calculated and measured average pressure drops, shown in Fig. 8.12 by the horizontal lines, is relatively small compared to the \( \Delta p \) oscillations during the cardiac cycle.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{fig8_12.png}
\caption{Temporal variation of pressure drop at the wall between ports 1 and 2 along a pulse cycle (Fig. 8.12a). Instantaneous pressure drops for parabolic spatial conditions are plotted along with \textit{in vivo} data obtained in a living dog. Also plotted is the time-averaged pressure. Fig. 8.12b is the measured inlet core velocity \( u_i \) (same plot as in Fig. 8.6a). Figs. 8.12a and b should be read in conjunction to calculate the phase angle.}
\end{figure}
During the acceleration part of the flow, the predicted peak pressure drop $-\Delta p_p$ is obtained at $t = 0.087$ s for the parabolic inlet conditions (Fig. 8.12a). From Fig. 8.12b, a peak Doppler velocity $u_{cp}$ is obtained at $t = 0.12$ s. This clearly indicates a phase lag between predicted $\Delta p_p$ and measured $u_{cp}$ that amounts to $\theta = -25^\circ$, since $u_{cp}$ lags predicted $-\Delta p_p$. At the beginning of diastole, flow reversal occurred ($u_{cl} = -7.7$ cm/s at $t = 0.24$ s). The peak in the predicted adverse pressure gradient $+\Delta p_p$ occurred at an earlier time so that the phase angle was $-37^\circ$. For the second smaller peak flow, $u_{cp}$ at $t = 0.33$ s, the phase angle was about $-27^\circ$. These calculated phase angles are within the range of $0^\circ$ to $-60^\circ$ known for physiological flows, and indicate a hysteresis effect between predicted $\Delta p$ peaks and measured $u_c$. Moreover, the predicted $\Delta p$ peaks appear to precede the measured $\Delta p$ peaks by phase angles of $-18^\circ$ to $-46^\circ$, progressively increasing during the cardiac cycle.

**Flow Calculation for Study 2**

Results of the numerical calculations where the measured pressure gradient $\Delta p(t)$ was used to calculate the flow field are shown in Figs. 8.13a and b. Fig. 8.13a shows the pulsatile Doppler blood flow mean velocity $u_c(t)$ and the spatial average instantaneous mean velocity, defined as

![Flow Diagram](image-url)
and centerline velocity \(u_c(t)\) obtained from the calculations. Also shown are the time-averaged velocities of the pulse cycle. Plotted below these results is the measured pressure drop \(\Delta p(t)\) (Fig. 8.13b). The phase angle \(\theta\) between the measured peak \(\Delta p\) and peak \(u_{c,p}\) signals was calculated as before. That is, if the peak \(\Delta p\) signal occurs at \(\omega t_1\), and the peak \(u_{c,p}\) signal occurs at \(\omega t_2 = \omega t_1 - \theta\), then since the circular frequency \(\omega\) is equal to \(\pi f = (2\pi/\tau)\), the phase angle \(\theta\) becomes \(360 \frac{(t_2 - t_1)}{T}\). Fig. 8.13a shows that during systole where flow acceleration occurs, at peak flow the mean \(\bar{u}_p\) is 50.8 cm/s at \(t = 0.152\) s, which is very near the Doppler velocity \(u_{c,p}\) (49.4 cm/s at \(t = 0.107\) s), and the phase angle \(\theta\) between \(\Delta p\) and \(-\bar{u}_p\) signals was about \(-35^\circ\) since \(-\bar{u}_p\) lags \(-\Delta p\). At the beginning of diastole, the adverse pressure gradient is peak \(+\Delta p\), flow reversal occurs, and the peak mean value \(-\bar{u}_p\) is \(-10.3\) cm/s at \(t = 0.293\) s, which is about 2.9 times larger than \(-u_{c,p}\) \((-3.58\) cm/s at \(t = 0.236\) s). The \(+\Delta p\), \(-\bar{u}_p\) phase angle increased to about \(-50^\circ\). For the second smaller peak flow during mid-diastole where flow acceleration occurred again, \(-\bar{u}_p \sim u_{c,p}\), similar to the value during systole. The \(-\Delta p\), \(-\bar{u}_p\) phase angle further increased to about \(-75^\circ\). The next cardiac cycle began before the mean \(\bar{u}\) decreased much at the end of diastole during the deceleration phase, presumably due to the large phase lag. Fig. 8.13a also shows that the ratio of predicted center line to mean velocity \(u_{c,p}/\bar{u}_p\) is less than the Poiseuille value of 2, being 1.5 at peak systole, 1.4 for peak reverse flow, and 1.8 for the second smaller peak flow.

In the physiological case, phase angles are near zero for peak flow (systole) and reverse flow, and positive for the second peak flow (mid-diastole). Values of \(\theta\) are positive because \(u_{c,p}\) occurred before \(\Delta p\).

Calculated phase angles are associated with the pressure gradient

\[
-\frac{\partial p(t)}{\partial z} = \frac{1}{A} \int_A u dA
\]

accelerating or decelerating the mean flow, i.e., \(Q(t) = \bar{u}(t) A\) including convective acceleration terms. Radial variation in pressure across the flow is negligible. The force balance on an element volume \(A dz\) in the axial direction \(z\) is

\[
-A dp - 2\pi r_w A \bar{u}_w dz = \rho A dA \bar{u}^2 dA + \rho \frac{\partial}{\partial t} Q(t) dz
\]

Thus, the pressure force must overcome the wall shear force to provide the net force necessary to provide convective and unsteady flow accelerations and decelerations. The calculated phase angles between measured \(\Delta p\) and predicted \(\bar{u}_p\) are nearly within the range of \(0^\circ\) to \(-60^\circ\) known for physiological flows. However, using the measured \(\Delta p\) as input, the calculated velocities, particularly during diastole, are out of phase and consequently not in agreement with the measured mean flow velocities. Flow reversal in femoral artery of a dog and phase lag between pressure gradient and flow have been reported by McDonald and his co-workers.\(^{17}\) In their case however, the agreement is much better between observed (high speed cinematography) and calculated velocities from a derived pressure gradient

\[
\frac{\partial p}{\partial z} = \frac{1}{c} \frac{\partial p}{\partial t}
\]

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where

$$\frac{\partial p_a}{\partial t}$$

is the time derivative of arterial pressure, and \( c \) is the recorded value for the peak to peak wave speed.  

Fig. 8.13a also shows the time-averaged mean velocity \( \bar{u}_a \) obtained by integration over the cardiac cycle along with the measured time integrated mean flow velocity \( u_{c,a} \). The value of \( \bar{u}_a = 16.9 \text{ cm/s} \) is about 12% larger than the Doppler value \( u_{c,a} = 15.1 \text{ cm/s} \), but the difference is relatively small compared to the velocity variations during the cardiac cycle.

In the calculations, the femoral artery of the dog was assumed to be rigid. Whereas the dynamic distensibility \( D \) or compliance \( c (= D/2) \) of the vessel wall was not measured, the measured arterial blood pressure \( P_a(t) \) at the iliac bifurcation was used to estimate the magnitude of wall movement during the cardiac cycle from

$$\frac{dr_w}{r_w} = \frac{1}{2} D d \left( P_a - P_a \right)$$

(8.17)

The mean blood pressure \( P_a = 72 \text{ mm Hg} \) and the peak systolic and minimum diastolic pressures were 92 and 52 mm Hg, respectively (Fig. 8.5). By using a value of \( D = 0.0013 \text{ 1/mm Hg} \) for the normal dog femoral artery,\(^1\) the systolic and diastolic excursions from the mean wall radius were estimated to be about the same, and the total excursion

$$\frac{\Delta r_{wd} + \Delta r_{wd}}{r_w}$$

was 2.6%. These estimated changes in lumen size during a heartbeat are not believed to significantly alter the findings of this investigation in which wall motion was neglected during systole where axial flow velocities are high. However, during early diastole, where flow velocities are low and flow reversal occurs, radial recoil of the vessel wall may induce axial velocities no longer negligible. In this case, the coupled equations of blood flow and wall motion would need to be considered.

### 8.6 Discussion

The numerical estimation of an instantaneous pressure drop for a pulsatile velocity or vice-versa is a challenging task. For blood flow, the non-Newtonian nature of blood viscosity poses an additional challenge in the calculation of the flow field. In the present case, temporal and spatial variations of shear rate and velocity add further complexity to the problem. Sharp changes in the instantaneous inlet boundary condition create numerical oscillations in the calculated data. In order to minimize these oscillations, the time-dependent inlet boundary condition is smoothened, as shown in Fig. 8.6a.

In the present analysis, numerical calculations are conducted for two consecutive pulse cycles in order to compare them and to obtain accurate results; the results for the second pulse cycle are calculated in continuation of the first one. Complete numerical data are reported only for the second cycle. Some important observations are noted as follows:

**Discussion for Study 1**

- Near peak systole, where sharp changes in velocity occur, less oscillation of pressure data are observed for the second cycle than the first cycle.
• The numerical calculation begins during the late accelerating phase of the systole, i.e., at about 0.05 s prior to the peak systolic velocity. Hence, the peak systolic pressure drop for the first cycle is meaningless and is ignored. When the peak pressure drop is compared between the second and third cycles, only a 0.13% change is observed.

• Determining the in vivo velocity pulse by non-invasive means such as the Doppler flow cuff allows the physiological pressure drop to be calculated. The in vivo prediction of pressure drop using an angiogram alone has a potential application from a medical diagnostic point of view, because it allows quantification of the physiological flow parameters in arterial blockages or stenoses.

Discussion For Study 2

• The problem is solved for three consecutive pulse cycles. It was observed that the results of the second and third cycles are within 1% of each other. It is evident that a repetitive solution is obtained from the second cycle and, therefore, the results for the second cycle are presented.

• For a practical scenario, the in vivo pressure measurements can be obtained using invasive means such as catheters. However, the pressure measured by a catheter needs to be modified in order to avoid the errors caused by the change in the pressure field due to insertion of a catheter.

8.7 Summary and Conclusions

The observations made in the present study may be summarized as follows.

Experimental Study

The velocity waveform for the femoral artery of a dog was tri-phasic with relatively small phase variations between the measured velocity and pressure drop at peak forward and reverse flow conditions. Hysteresis effects were observed during the flow acceleration and deceleration phases of the cardiac cycle.

Numerical Study

Summary and Conclusions for Study 1

1. Determining the in vivo velocity pulse by non-invasive means such as the Doppler flow cuff allows the physiological pressure drop to be calculated.

2. The calculation of instantaneous pressure drop across a segment of the femoral artery of a dog was found to be in reasonable agreement with the measured in vivo pressure drop during the cardiac cycle. The shape of the inlet velocity profile was assumed to be parabolic since the femoral segment was deep in the circulation and branch-free upstream (pressures were measured in ligated branches). Numerical calculation of the time-averaged pressure drop over the cardiac cycle was about 10% higher than the measured in vivo value, which could also be estimated by using the mean flow Poiseuille relation with a momentum correction for the vessel taper.

3. A phase lag between predicted peak $\Delta p$ and measured peak Doppler velocity $u_c$ at systolic peak flow, early in diastole, and in mid-diastole, was calculated in the range of phase angles $\theta$ of $-25^\circ$ to $-37^\circ$. The predicted $\Delta p$ peaks preceded the measured $\Delta p$ peaks at peak systolic flow, early in diastole, and in mid-diastole by phase angles of $-18^\circ$ to $-46^\circ$.

4. For the parabolic inlet condition, the mild taper of the artery caused some changes in both shear rate and stress; specifically, a higher magnitude of oscillating shear rate at the wall during both systole and diastole was observed downstream. Furthermore, the steeper gradient of wall shear rate due to its taper affected not only the non-Newtonian viscosity of blood and the shear stress, but also the pressure.
Summary and Conclusions for Study 2

1. A progressive increase in phase lag between the inlet pressure gradient and calculated mean flow was predicted. The phase lag increased from a few degrees during early systole to about $-75^\circ$ during late diastole. A similar result has been reported by McDonald and his co-workers.$^{17}$ Their results show an increase of phase lag from zero degrees during early systole to about $-58^\circ$ during late diastole.

2. The time-averaged mean velocity $\overline{u}_a$ obtained by integration over the cardiac cycle is found to be about 12% larger than the measured time-integrated Doppler flow velocity $u_{ac}$. 

3. The magnitude of the measured peak velocities $u_c$ is found to be in fair agreement with the calculated mean velocities $\overline{u}_a$. This fact is clearly evident when the systolic part of the pulse cycle and the time-averaged values are compared. This observation can also be made with caution in the diastolic region. In the diastolic region, the calculated phase difference is comparatively higher than the experimental observations, resulting in apparent disagreement in the instantaneous velocities. This needs to be investigated further from the perspective of viscoelastic wall effect.

From the comparisons of numerical Studies 1 and 2, we conclude that to estimate the pressure drop–flow rate relationship in the mildly compliant femoral artery wall of the dog by using rigid wall theory, it may be better to use the measured velocity than the measured pressure gradient as input into the calculation. Hence, it is essential to use an accurate temporal and spatial distribution of velocity inlet profile for the calculation in order to obtain an accurate pressure distribution. An earlier study$^{12}$ has suggested this approach, i.e., used the measured flow rate to estimate the mean wall shear stress in an elastic artery model. Our study determined that by using the measured Doppler flow velocity as input, the calculated $\Delta p$ is in fairly good agreement with the measured $\Delta p$, but is still somewhat out of phase. On the other hand, using the measured $\Delta p$ as input, the calculated velocities, particularly during diastole, are more out of phase and not in agreement with the magnitude of the measured flow velocities.

It is believed that the calibrated Doppler flowmeter provides reliable flow velocity measurements. The very high frequency excitation signal provides nearly instantaneous Doppler frequency shift data even with an internal low pass filter as discussed in the Experimental Method section above. The validyne differential pressure transducer has a high frequency response, and, with calibration, also accurately measures the instantaneous $\Delta p$. Therefore, the physiological phase difference between the $\Delta p$ and $u$ signals are shown in Fig. 8.5 and in the $\Delta p$–$u$ hysteresis curve in Fig. 8.7. Reproduction of physiological measurements such as these is a challenging chore for computational fluid mechanics.

In vivo measurements in the laboratory have repeatedly shown that the velocity pulse and the pressure drop are not only dependent on the physiological location of the artery, but also vary from one individual dog to another. The results reported in earlier work$^6$ are for a femoral artery of a different dog with a similar trend of measured instantaneous velocity but with a different magnitude. Hence, a unique set of results for any dog, or, in other words, a generalization of results, is practically impossible. This study has attempted to propose a method to calculate pressure and velocity changes in an artery using an arterial angiogram, the non-Newtonian viscosity of blood, and an instantaneous velocity or pressure pulse. In conclusion, the hemodynamic measurements reported herein are believed to provide a set of consistent data that may be useful in subsequent studies of arterial flows and numerical flow calculation methods. Clearly, more work is needed to acquire a better understanding of arterial flow and pressure variation along vessels during the cardiac cycle.

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**Nomenclature**

\[ A = \text{cross-sectional area} \]
\[ D = \text{distensibility} \]
\[ c = \text{compliance} \]
\[ d = \text{lumen diameter} \]
\[ l = \text{length of the vessel segment} \]
\[ n_i = \text{normal vector} \]
\[ p = \text{pressure} \]
\[ p_1 = \text{wall pressure at reference location (initial node)} \]
\[ p_i = \text{wall pressure at any downstream nodes} \]
\[ Q = \text{flow rate} \]
\[ r_w = \text{lumen radius} \]
\[ s_i = \text{stress vector} \]
\[ t = \text{time} \]
\[ T = \text{period of heart beat} \]
\[ u_i = \text{velocity vector} \]
\[ z = \text{distance along the main lumen} \]
\[ \alpha = \text{dimensionless frequency parameter, or Womersley number} \]
\[ \dot{\gamma} = \text{shear rate} \]
\[ \varepsilon = \text{penalty parameter} \]
\[ \eta = \text{viscosity} \]
\[ \theta = \text{phase angle between } \Delta p \text{ and } u \]
\[ \nu = \text{kinematic viscosity } (\eta/\rho) \]
\[ \rho = \text{density} \]
\[ \sigma_{ij} = \text{stress tensor} \]
\[ \omega = \text{circular frequency, } 2\pi/T \]
\[ \tau_w = \text{wall shear stress} \]

**Subscripts**

\[ a = \text{time-averaged} \]
\[ c = \text{Doppler measurement} \]
\[ cl = \text{centerline condition} \]
\[ p = \text{peak value} \]
\[ \Delta = \text{difference} \]
\[ * = \text{numerically computed value} \]

**Superscripts**

\[ (-) = \text{spatial average} \]
References