ESTIMATION OF SUBJECT-SPECIFIC HEMODYNAMIC PARAMETERS USING DIFFERENTIAL EVOLUTION ALGORITHM AND PARALLEL COMPUTING

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Abstract— Cardiovascular system modeling involves a great number of parameters (resistances, compliances, network geometries, etc.) which are unknown a priori and need to be identified. The present work aims at developing an identification procedure for estimating subject-specific hemodynamic parameters based on a one-dimensional (1-D) tree-like vascular flow model. For the present identification method, the arterial stiffness and stress-free lumen radius as well as the terminal lumped resistances and compliance were estimated. This work presented a preliminary validation using in vivo data to assess the feasibility and accuracy of the proposed method. The results show that the identification method is accurate and feasible. An excellent fit was achieved with root-mean-square error (RMSE) of 2.18% between the in vivo measured and the model produced pressure waveforms.

Keywords— Windkessel models, reflection coefficient, hemodynamic inverse problem, parameter identification, least-squares estimation.

I. INTRODUCTION

To date, our knowledge of human circulatory physiology has been confined in the realm of the forward or direct (modeling) problem; namely, for any given vascular geometry and vessel wall properties, one can derive or assess the flow rate or pressure waveforms subjected to some specified inflow conditions [1-3]. Contrary to these direct problems, the conjugate inverse (diagnosis) problem, which solves for the physiologic or pathophysiologic parameters for a given observable measurements obtained at some vascular sites, is of much greater clinical value [4].

Historically, models employed to solve the inverse problem are the Windkessel models connected to a 1-D tubular conduit and were solved using the frequency domain analysis. However, under this simplified model framework, it was concluded that no unique solution to the inverse problem exists for a specified inflow impedance information [5]. This conclusion, somehow, discourages the intention of developing a cardiovascular parameter identifier.

The key to solving the inverse problem is to take the spatial vessel distribution into account to better represent the underlying wave propagation, reflection, and re-reflection phenomena in topological tree-like vasculature [6], and then basing on this truer vascular representation to develop the related inverse solution method. Recently, an identification method that estimates distributed arterial mechanical properties has been proposed [7]. It was developed to solve the inverse problem by building on a wave propagation model. The fitting procedure, based on local sensitivity indices and fitting rules, is used to estimate the individual model parameter set that yields the best fit between the measured and simulated data. However, local and global sensitivity analyses [7], [8] have indicated that the relationship between the model parameters and the predicted output (i.e., pressure and flow) is quite complex because the model output is significantly influenced by multiple system parameters. Therefore, with this approach, it is not easy to find a unique parameter set that absolutely corresponds to the global minimum of the predetermined objective function.

The present work proposes an identification procedure for estimating subject-specific hemodynamic parameters based on a one-dimensional (1-D) tree-like vascular model. The proposed identification methods intend to solve the hemodynamic inverse problem to yield the estimation of model parameters which are of clinical interest. Modern computational fluid dynamics, wave mechanics and stochastic state estimation and parameter identification theories that have been well
developed in aerospace and mechanical engineering will be adopted in the present method development.

II. METHODOLOGY

A. Governing Equations for One-Dimensional Blood Flow

The blood flow in a tubular duct has been modeled by taking the cross-sectional average of the flow variables represented by Navier-Stokes equations in three dimensions, with the assumption that the flow is incompressible, Newtonian, axisymmetric, and laminar. Besides, pressure is a function of cross-sectional area only and the velocity profile is flat but with a boundary layer of thickness next to the wall. The resulting one-dimensional (1-D) equations in the longitudinal direction can be expressed as [9], [10]:

\[
\frac{\partial Q}{\partial t} + \frac{\partial F}{\partial x} = S
\]

where \( A \) is the lumen cross-sectional area of the tube with radius \( r \), \( q \) is the flow rate, \( \rho \) is the blood density, \( \nu \) is the kinematic viscosity (assumed to be constant), \( \delta \) is the boundary layer thickness (assumed to be constant), and \( p \) is the static pressure. The independent variables \( x \) and \( t \) represent, respectively, the spatial and time coordinates of the postulated 1-D governing equations.

To close up the above flow equations having three state variables (\( A, q, \) and \( p \)), an inclusion of a third equation, namely the state equation of the tube structure:

\[
p(x,t) - p_0 = \frac{4}{3} \frac{E h}{A_0(x)} \left[ 1 - \sqrt{\frac{A_0(x)}{A(x,t)}} \right]
\]

where \( p_0 \) is the ambient or tissue pressure, \( A_0 \) is the cross-sectional area with radius \( r_0 \) at zero transmural pressure (i.e., at \( p = p_0 \)), and \( E \) and \( h \) are the Young’s modulus and thickness of the vascular wall, respectively.

B. Arterial Network Model

The human arm arterial network comprising five vessel branches (\( N=5 \)) with two bifurcations and three terminal ends (\( M=3 \)), as shown in Fig. 1, was employed in this work for hemodynamic simulations and parameter estimation.

Fig. 1. Procedure of pressure-flow rate \((pq)\) method in parameter identification. Flow rate measured at the inflow end is taken as the input function and pressure at observational site is used as the reference to be compared with the model-produced pressure reconstruction in the optimization procedure. \( J(\theta) \) is the cost function, \( \Theta_{i,G+1} \) is the model parameter set corresponding to the \( i \)-th population and the \( G \)-th generation.

1) Outflow Boundary Conditions: In practical clinical applications, the 1-D flow modeling of an arterial tree structure has to be terminated at certain finite number of bifurcation generations. Windkessel models [11], lumped models [12], [13], and structured tree models [9], [10], [14] have been used to represent the overall effect contributed by the truncated downstream vasculature bed. A general formalism of characteristic outflow boundary conditions for coupling 1-D terminal ends and Windkessel models has been developed in [15], [16]:
where the subscript in $z_i$ represents the $i$-th wave mode propagating with characteristic velocity $\hat{\lambda}_i$, $R_i$ is the reflection coefficient defined as $R_i = (R^i - Z_n) / (R^i + Z_n)$, $c$ is the wave speed, $R^i$ is the terminal resistance of the $i$-th element, $C$ is the terminal compliance, and $Z_n = \rho c_0 / A_n$ is the characteristic impedance of the connected 1-D tube. This new form of boundary condition representation is particularly useful in the study of vascular hemodynamics, and is thus adopted in this work for outflow boundary treatment.

2) Numerical Solver: Numerical solutions for the arterial network model are obtained by using the validated high-resolution scheme in [17] to achieve high-order accuracy in terms of having small numerical dissipation and dispersion errors.

3) Model Parameter: The arterial network model has vessel-related parameters ($L$, $r_0$, and $Eh$) in each 1-D branches governed by Eqs. (1)-(3) and Windkessel-related parameters ($R^i$, $\hat{\lambda}_i$, and $C$) in the terminal model represented by Eq. (4). These system parameters should either be known a priori or be identified. Sensitivity analysis [8] has shown that vascular length $L$ least influences the output. In the present study, $L$ has been initialized and specified using the geometrical image data. After parameter initialization, the final unknowns to be determined can be defined into a model parameter set:

$$\theta = \{Eh, R^i, \hat{\lambda}_i, C, R_j^i\}_{i=1}^{N} \cup \{R_j, \hat{\lambda}_j, C, R_j^i\}_{j=1}^{M},$$

where the indices $i$ and $j$ denote the $i$-th vessel branch and the $j$-th terminal end, respectively. The 1-D arterial network model possesses at least $2N+3M$ parameters that should be estimated.

C. Parameter Identification Procedure

The schematic showing the identification procedure using pressure-flow rate ($pq$) data pairs, namely $pq$ method in this work, is illustrated in Fig. 1. Flow rate and pressure waveforms at the inflow end are taken as the system input and observer, respectively. Random initial system parameter guess initialized the differential evolution (DE) search process until the objective function was attained within a predetermined error bound.

1) Least-Squares Estimation: Suppose that the nonlinear relationship between observations, model states, and parameters at the discrete sampling time $k$ are related through the observer model:

$$z_k = h(Q_k, \theta) + v_k$$

where $z$ is the measurement vector, $h$ is the nonlinear function, $Q$ is the assembly of all the 1-D state variables (defined in Eq. (2)) assigned at each cell center of the discretized vessel, $\theta$ is the parameter set with dimension $n$, and $v$ is the measurement errors.

Weighted least-squares estimation is used to find the most probable estimate $\hat{\theta}$ based on the measured samples $z$ such that the weighted sum of the squared error of the data fit is minimized. Thus, the task is to minimize the following cost function [18]:

$$J(\theta) = \frac{1}{2} \sum_{k=1}^{N} (z_k - \hat{z}_k)^T W_k (z_k - \hat{z}_k)$$

where $\hat{z}$ is the model-predicted output and $W$ is the predetermined weighting matrix.

2) Differential Evolution (DE) Algorithm: The proposed process of parameter identification is performed using the differential evolution (DE) algorithm proposed by Storn and Price [19]. The DE algorithm, a stochastic global search method, is adopted herein because it can handle non-differentiable and nonlinear cost functions, and is simple to implement and conveniently amenable to parallel computing. DE search can be initialized with a randomly generated initial guess, covering the entire parameter space broadly defined in the possible physiological range.

The validated hemodynamic measurements, some used as the inflow condition and the rest taken as the observer $z$ defined in Eq.(6), are fed into the model-based identification procedure. The goal is to find the most fitted system parameters that minimize the cost function of Eq. (7). This is accomplished using the DE search algorithm, starting with a randomized initial population and then applying mutation, crossover, and selection operations from generation to generation going through the DE optimization until the cost function is minimized within a predetermined error bound, as illustrated in Fig. 2.
workstation with \( n \) cores is shown in Fig. 3. All sets of parameters (population number = \( N_p \)) searched by the DE algorithm are evenly distributed over the cores via the MPI_Scatter call for 1-D flow simulation. After the cost function has been independently evaluated, the MPI_Gather call is used to collect them. This framework is easy to implement without the necessity to change the original 1-D code. The speedup performance is shown in Fig. 4.

Fig. 2. Illustration of differential evolution (DE) search algorithm for an example of 5 parameters. The indices \( i \) and \( G \) denote the \( i \)-th population and the \( G \)-th generation, respectively. The superscript \( M \) and \( C \) represent, respectively, the mutant and crossover vector.

Fig. 3. Parallel computing paradigm. MPI: message passing interface; MPI_Scatter: distributes distinct messages from a single source task to each task in the group; MPI_Gather: gathers distinct messages from each task to a single destination. This routine is the reverse operation of MPI_Scatter. \( \theta_i \) is the vector of the model parameter set with \( N_p \) population size.

### III. RESULTS AND DISCUSSION

For assessing the identifiability and feasibility of the inverse problem using the presently proposed model-based identification algorithm, a method validation with \textit{in vivo} data is presented. The \textit{in vivo} data were taken from Leguy et al. [7], which were digitized into discrete time-series for the present model validation. The flow rate is served as the input function and system parameters are tuned so as to achieve a best fit of the pressure waveform by minimizing the predetermined cost function. The fitting result is shown in Fig. 5. An excellent fit is achieved with root-mean-square error (RMSE) of 2.18% between the \textit{in vivo} measured and the model produced pressure waveforms at the inlet of artery No. 1. The parameters identified by the present \( pq \) method and by the reverse method are displayed in Table I. Arterial Young’s modulus estimates obtained for the artery No. 1 using the present \( pq \) method and those assessed by pulse wave velocity (PWV), distensibility, PU-loop, and reverse methods are compared in Fig. 6. Note that much larger data scatter appears in the results obtained by PU-loop and distensibility methods. It is suspected that great uncertainty due to the repeatability and reproducibility problems are associated with these methods when practically performed in the clinical setting.
The Young’s modulus of the artery No. 1 was well estimated using the proposed \( pq \) method, as demonstrated by comparing to the results of the reverse method shown in Fig. 6 and Table I. The present method and the reverse method developed by Leguy et al. [7] were both based on the 1-D flow model. Both methods yield very agreed identification results, providing more confidence in vascular stiffness evaluation as compared to the previous PWV, distensibility and PU-loop methods.

The differences in these two 1-D flow-based methods lie in the numerical algorithm used for solving the 1-D equations and the identification procedure for estimating the model parameters. The present method uses the high-resolution Roe’s scheme [17] and the stochastic global search DE optimizer [19] whereas the reverse method uses a spectral element method in conjunction with local sensitivity indices [7]. For the local sensitivity indices method to converge, a good initial guess of each starting parameter is required. For example, the reverse method uses the average of the estimates obtained by the distensibility and PWV methods to assist the initial guess and requires four state variables at three measurement locations to be provided in its estimation process. In contrast, the present approach uses a randomly generated initial guess to start with and only demands data be supplied at one point. In this way, the proposed method would have greater potential on the future clinical applications.

![Figure 5](image)

**Fig. 5.** Fitting results between measured (solid line) and model reconstructed (dashed line) pressure waveforms at the inlet of artery No. 1. The root-mean-square error (RMSE) values were computed using simulated and measured waveforms and are expressed as percentages relative to the *in vivo* values.

![Figure 6](image)

**Fig. 6.** Young's modulus estimates obtained for the artery No. 1 using pulse wave velocity (PWV), distensibility, PU-loop, reverse, and \( pq \) methods. Error bars show the measurement uncertainties for the PWV, distensibility, and PU-loop methods. Data are digitized from Leguy et al. [7] and used in the present plots.

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<th>Parameter</th>
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<th>Reverse method</th>
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<td>( R^2 )</td>
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\( r_0 \) (cm): stress-free lumen radius
\[ E \text{ (MPa)} : \text{Young's modulus} \]
\[ R^*_f : \text{reflection coefficient} \]
\[ C \text{ (ml/mmHg)} : \text{peripheral compliance} \]
\[ R_f \text{ (mmHg s/ml)} : \text{peripheral resistance of the 2nd element} \]
NA: not available

IV. CONCLUSIONS

A model-based identification algorithm was developed here to solve the inverse problem for estimating the hemodynamic parameters of the arterial system. This work presented a preliminary validation using in vivo data to assess the feasibility and accuracy of the proposed methods. The results show that the proposed identification method is accurate and feasible. For identifying subject-specific parameters pertaining to a segmental arterial network of an arm, agreed hemodynamic waveforms reconstructed by the model were compared favorably to the in vivo measurements. This approach may provide researchers and clinicians with a deeper mechanistic understanding of the genesis of diseases in major vessels and eventually, as aided by our knowledge in medicine, lead to a better cardiovascular monitoring or diagnostic tool.

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