A Nonlinear Model of the Arterial System Incorporating a Pressure-Dependent Compliance

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Abstract—The three-element modified Windkessel model has been widely used to study the characteristics of the systemic arterial system. This model provides most of the features of the systemic input impedance, but does not describe the nonlinear effect of the pressure dependence of arterial compliance. The current investigation examines the hemodynamic consequences of such an inclusion. Simultaneous aortic pressure and flow during control and brief descending aortic occlusion were measured in open chest anesthetized experimental dogs. A numerical procedure was implemented to compute constant compliance linear and nonlinear compliance model-predicted pressure waveforms with flow as the input. Results show that the nonlinear compliance model in general can more accurately predict the measured pressure waveforms during control and during acute pressure loading. The difference between the predicted waveforms is more pronounced when blood pressure is high and when the pulse pressure is large.

INTRODUCTION

The Windkessel model of the arterial system was established about a century ago for the calculation of stroke volume [1], a then recognized parameter that was considered pertinent for assessing ventricular pumping ability. The model, however, lacked pulse transmission characteristics [2]. Decades later, additional models of the arterial system emerged [3]. They are mostly based on the assumption of a constant arterial compliance.

For many years, the characteristics of the arterial system were studied by applying a modified three-element Windkessel model in which the systemic arterial tree is analogued with the compliance of the arteries, the resistance of the peripheral vessels, and the characteristic impedance of the proximal aorta. Among the characteristics investigated, changes in compliance of the aorta have attracted the most attention. These alterations are closely related to the mechanical properties of the vessel wall and are linked to certain cardiovascular diseases such as aging, atherosclerosis and hypertension [4], [5]. One approach to obtain the aortic compliance, derived from the Windkessel model, is to compute the diastolic aortic pressure decay time constant [6], [7]. Although it has been known for some time that the total arterial compliance decreases with increasing pressure [8]-[10], the aortic compliance is still considered a constant value in these model-based calculations. The present paper examines the consequences of incorporating a pressure dependent compliance in a modified arterial system model. This nonlinear model is evaluated under control and acute pressure-loading conditions.

THEORETICAL ANALYSIS

The proposed nonlinear model incorporating an arterial compliance that is a function of pressure \( C(P) \) is shown in Fig. 1. \( Z_o \) and \( R_s \) represent the characteristic impedance of the ascending aorta and the peripheral resistance, respectively.

If the pressure associated with a given aortic compliance \( C_s(t) \) is \( P(t) \), then the relation between \( C_s \) and \( P \) can be expressed as follows:

\[
C_s(P) = a * e^b(P(t))
\]

(1)

where \( a \) and \( b \) are constants determined from curve-fitting of experimentally measured aortic pressure and diameter reported previously [9], [10]. The least-square fit has a correlation coefficient of \( r > 0.97 \).

The flow through the compliance branch can be written as

\[
Q_c(t) = Q(t) - P(t)/R_s
\]

(2)

where \( Q(t) \) is the measured aortic flow.

Furthermore, it is clear that

\[
Q_c(t) = C_s(P) * dP(t)/dt.
\]

(3)

Equations (2) and (3) describe the flow in the same branch of the circuit. They can be equated leading to the following expression:

\[
dP/dt = (Q(t) - P(t)/R_s)/C_s(P).
\]

(4)

This equation defines the relationship between aortic pressure and flow for the nonlinear aortic compliance. Equation (4) can be solved numerically, noting that

\[
\Delta t = t_{i+1} - t_i \equiv dt
\]

(5)
The characteristic impedance of the aorta was determined in the time domain from the upstroke of aortic pressure and flow during the first 60 ms of the systole (11), i.e.,

$$Zo = \frac{(P - Pd)}{Q}$$

(9)

where $Pd$ is the diastolic aortic pressure. Data were sampled at 10 ms intervals. The instantaneous ratio of $(P - Pd)/Q$ was plotted for the first six pairs of sampled pressure and flow data and the average value of this ratio was obtained as $Zo$. The peripheral resistance was calculated from the ratio of mean pressure to mean flow

$$Rs = \frac{\bar{P}}{\bar{Q}}.$$

(10)

The time constant is obtained by fitting the diastolic portion of the aortic pressure to a monoexponential decay curve. The correlation coefficient ($r$) was generally greater than 0.93. The heat rate was calculated from the inverse of an average of several $R - R$ intervals of the ECG.

Since the model is based on the nonlinear dependence of compliance, the determination of $a$ and $b$ in (1) requires a special procedure. For the current analysis, pressure volume data is required to determine $C(P)$, from the definition

$$C = \frac{dV}{dP}.$$

(11)

The strategy used is shown by the flow-chart in Fig. 2. $Rs$, $Zo$, and $t$ are input to the model as previously determined constants. Aortic flow is obtained from experimental data; $a$, $b$ initial values are based on curve-fitting result, and the initial value of $P(t)$ is from measured end diastolic pressure. The next step is to solve the difference (6) and (7). As the result, the aortic pressure based on the nonlinear model is predicted. In order to evaluate the performance of the model, the error $E$ is defined as

$$E = \sqrt{\frac{N}{\sum_{i=1}^{N} \left(P \text{ calculated } (t_i) - P \text{ measured } (t_i) \right)^2}}.$$

(12)

The set of parameters including $a$, $b$ and the initial value of $P(t)$ are adjusted until $E$ meets the desired error within
physiological limits. The nonlinear model then outputs aortic $P$, aortic compliance, as well as the pressure associated with the nonlinear compliance for later plotting.

RESULTS

Simultaneously measured aortic pressure and flow waveforms at control and during a brief descending aortic occlusion are shown in Fig. 3. The increase in pulse pressure and the steeper decline in diastolic pressure are visible.

Table I lists the measured and calculated hemodynamic parameters for both control and aortic occlusion. There is a slight change in the calculated characteristic impedances between control and aortic occlusion. The peripheral resistance is significantly elevated during aortic occlusion. Compliance calculated from the diastolic time constant is decreased, as expected, during aortic occlusion. The rapid aortic pressure decay after valve closure gives rise to a shortened time constant.

The pressure waveforms predicted by the linear model and the nonlinear model are compared to the measured pressure waveform during control as shown in Fig. 4. There is little difference among them at the upstroke of early systole. The differences are pronounced at midsystole, and more so in late systole. The difference at the systolic peak is about 9% for the linear model and 3% for the nonlinear model. There is a little phase shift between the nonlinear model predicted pressure and the actual measured pressure. The linear model in general has the most error when the flow rate is high and when the pressure is also high. The nonlinear model in general predicts the overall pressure waveform better. The compliance change is relatively small, about 5% throughout the cardiac cycle (Table II).

Fig. 5 shows the predicted waveforms as compared to the measured waveform during acute pressure loading. The discrepancies between the linear model and measured waveform are larger as compared to control. The nonlinear model whose compliance can adjust to the change in pressure predicts the measured waveform relatively well. The compliance changes within the cardiac cycle well exceeded 10% (Table II).

DISCUSSION

Characteristics of the arterial system can usually be obtained from the input impedance of the arterial tree. The input impedance is useful because it only requires the simultaneous measurements of the pressure and flow waveforms at the ascending aorta to provide information regarding the interaction between the proximal aorta and the peripheral vascular beds. In addition, certain pulse wave transmission characteristics can be inferred. Although distributed models can more accurately predict the propagating pressure and flow waveforms and the input impedance, they are generally more complex and time consuming for individual parameter identification which often outweigh any additional information that can be obtained. Lumped models of the arterial tree, therefore, were proposed. Among them, the three-element Windkessel model [20] has been the most popular. The main reasons for this are that it can predict the gross features of the input impedance [2], [3]. The inertial term is neglected in this model because its contribution has been found to be small.

The three-element Windkessel model is based on the linear pressure-volume relation incorporating an elastic modulus that is pressure independent. This assumes that the compliance of the arterial system remains constant throughout the cardiac cycle. This is not exactly correct.
TABLE I
MEASURED AND COMPUTED HEMODYNAMIC PARAMETERS FOR THE LINEAR MODEL OF THE ARTERIAL SYSTEM (Mean ± SEM)

<table>
<thead>
<tr>
<th></th>
<th>HR (1/min)</th>
<th>P_s (mmHg)</th>
<th>P_d (mmHg)</th>
<th>Z_o (mmHg/ml/s)</th>
<th>R_s (mL/mmHg)</th>
<th>( \tau ) (s)</th>
<th>C_t (mL/mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>125.3 ± 2.8</td>
<td>116.3 ± 5.6</td>
<td>91.7 ± 3.5</td>
<td>0.25 ± 0.09</td>
<td>3.1 ± 0.8</td>
<td>1.1 ± 0.06</td>
<td>0.48 ± 0.12</td>
</tr>
<tr>
<td>Occlusion</td>
<td>120.0 ± 2.5</td>
<td>149.0 ± 5.8</td>
<td>104.3 ± 5.7</td>
<td>0.36 ± 0.07</td>
<td>6.35 ± 1.16</td>
<td>0.89 ± 0.06</td>
<td>0.22 ± 0.06</td>
</tr>
</tbody>
</table>

HR = heart rate; \( P_s \) = systolic pressure; \( P_d \) = diastolic pressure; \( Z_o \) = characteristic impedance of the aorta; \( R_s \) = peripheral resistance; \( \tau \) = diastolic aortic pressure decay time constant; \( C_t \) = arterial compliance, constant throughout the cardiac cycle.

Previous studies on pressure-strain elastic modulus [9], [10], [12]

\[ Ep = (\Delta p / \Delta D) \bar{D} \]  

where \( Ep \) is the pressure-strain elastic modulus. The relationship was derived from an optimization procedure, based on the compliance estimation from the diastolic aortic pressure decay time constant. The data in general show a tendency of the compliance to decrease with an increasing pressure, although in some of the fitted curves compliance actually increases with pressure even when the mean pressures are within the physiologic range. This was not observed in the present study.

Liu et al. [15] made a comparison of arterial compliance values obtained by different methods, including pressure-dependent compliances. Our in vivo data from the dog aorta are in good agreement with their in vivo results.
findings in humans if body size is used as a scale factor. However, their in vitro compliance values of excised human arteries are smaller.

The current analysis does not differentiate large vessel compliance from those of peripheral vessels as that shown by Finkelstein et al. [16]. It is known, however, that a large portion of the arterial compliance resides in the proximal portion of the aorta. Peripheral arteries account for less than 5% of the total arterial system compliance. The aorta with little viscous losses is much more elastic than peripheral vessels. Thus, such pressure dependence of compliance has its contribution more from the aorta than from the peripheral arteries.

The decreasing compliance with an increasing pressure reflects the arterial wall components behaviors under normal and pressure loading conditions. It has been shown that the distensibility of the blood vessel is dependent on the degree of the smooth muscle activation [17], [18], the elastic behaviors of the elastic laminae and the collagen fibers. In vitro studies with excised blood vessels have shown that the elastic moduli of these components increase with an increasing pressure. It is not clear, however, from the current nonlinear model prediction whether such changes in compliance is passive or active.

The study of the pressure dependent behavior of arterial compliance is important, because of its relevance to many cardiovascular diseases. In hypertension, for instance, the increased pressure is always associated with a reduced compliance. Reduced compliance impedes ejection and is detrimental to normal left ventricular function [19]. It is not clear, however, whether an increased pressure is due to a decreased compliance as a result of alterations in vessel wall properties, or that a decreased compliance is a consequence of an elevated arterial pressure. At least in the current investigation in acute occlusion of the aorta, the decreased compliance is more likely due to an increase in pressure. In the case of chronic hypertension due to atherosclerosis, such a decrease in compliance may be manifested through a change in vessel wall properties.

The present studies, however, conclude that a constant compliance model or the linear three-element windkessel model is useful when pressure variation within the cardiac cycle is small and at about normal mean blood pressure. When the mean blood pressure is high, or when the pressure variation within a single cardiac cycle is large, the nonlinear pressure dependent compliance model proposed here will more accurately reflect the behavior of the arterial system.

REFERENCES


Ting Cui (S'89) was born on June 24, 1957. She received the B.S. and M.S. degrees in precision instrument engineering from Tianjin University, China, in 1982 and 1984, respectively. She received the Ph.D. degree in biomedical engineering from Rutgers University, Piscataway, NJ in 1990. Her research interests include cardiovascular system modeling and simulation, microprocessor control of cardiac assist device, and data acquisition and analysis of physiological signals.

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